

ABSTRACT

OF

The Proceedings of the Fortieth
Annual Meeting of the Association
of Life Insurance Medical
Directors of America

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VOL. XVI

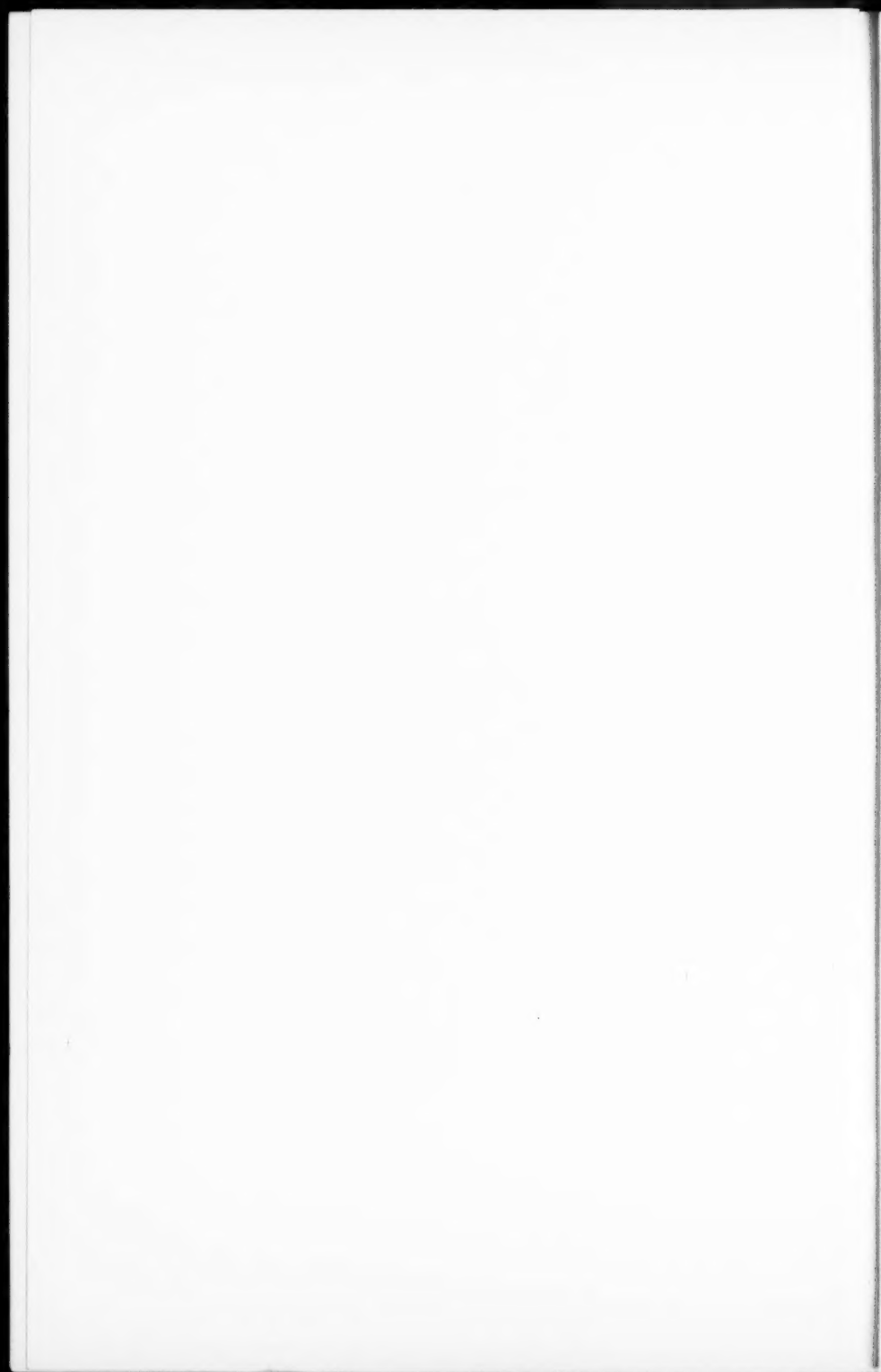
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MEDICAL DIRECTORS OF AMERICA

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vii

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An Abstract of the Proceedings

OF THE

Association of Life Insurance Medical Directors of America

FORTIETH ANNUAL MEETING.

The Fortieth Annual Meeting of the Association of Life Insurance Medical Directors of America was held at the Home Office of the Prudential Insurance Company, Newark, New Jersey, on October 24 and 25, 1929. The Association was the guest of the Prudential Insurance Company. President J. Allen Patton was in the chair.

The following members and delegates were present at some time during the sessions: G. E. Allen, C. D. Alton, E. M. Armstrong, W. B. Aten, A. E. Awde, H. A. Baker, W. B. Bartlett, J. T. J. Battle, W. W. Beckett, E. W. Beckwith, C. D. Bennett, C. C. Berwick, E. B. Bigelow, C. C. Birchard, W. F. Blackford, J. T. Bowman, F. G. Brathwaite, C. T. Brown, B. F. Byrd, W. B. Carpenter, L. D. Chapin, C. L. Christiernin, C. P. Clark, H. W. Cook, P. M. Cort, D. B. Cragin, J. L. Crawford, H. C. Cruikshank, George Cullen, R. M. Daley, E. J. Dewees, E. G. Dewis, E. S. Dillon, H. W. Dingman, P. G. Drake, O. M. Eakins, J. E. Engelson, C. H. English, W. G. Exton, H. H. Fellows, R. A. Fraser, H. M. Frost, F. I. Ganot, H. W. Gibbons, A. H. Gordon, Angus Graham, R. J. Graves, J. B. Hall, W. F. Hamilton, Frank Harnden, J. D. Heacock, C. A.

Heiken, A. B. Hobbs, J. P. Honsberger, J. C. Horan, Ross Huston, J. P. Hutchinson, W. G. Hutchinson, Lefferts Hutton, C. B. Irwin, W. A. Jaquith, B. Y. Jaudon, F. L. B. Jenney, A. O. Jimenis, A. E. Johann, G. E. Kanouse, J. E. Kinney, A. S. Knight, W. P. Lamb, J. M. Livingston, M. T. McCarty, N. C. McCloud, F. H. McCrudden, C. B. McCulloch, L. F. MacKenzie, W. T. McNaughton, F. W. McSorley, H. L. Mann, W. L. Mann, E. L. Mathias, O. F. Maxon, J. C. Medd, H. L. Metcalf, W. F. Milroy, J. T. Montgomery, R. C. Montgomery, William Muhlberg, C. T. Necker, Herbert Old, M. I. Olsen, B. W. Paddock, G. W. Parker, J. A. Patton, J. S. Phelps, C. B. Piper, J. E. Pollard, J. J. Post, W. W. Quinlan, W. A. Reiter, F. P. Righter, A. J. Robinson, T. H. Rockwell, O. H. Rogers, E. K. Root, R. L. Rowley, C. L. Rudasill, S. C. Rumford, E. F. Russell, H. C. Scadding, C. E. Schilling, S. B. Scholz, J. T. Sheridan, D. M. Shewbrooks, J. L. Siner, Dewitt Smith, J. M. Smith, M. K. Smith, Morton Snow, H. F. Starr, J. B. Steele, L. G. Sykes, B. C. Syverson, W. E. Thornton, F. L. Truitt, J. P. Turner, C. A. Van Dervoort, W. R. Ward, W. E. H. Wehner, F. S. Weisse, F. L. Wells, D. E. W. Wenstrand, S. S. Werth, C. F. S. Whitney, T. H. Willard, R. L. Willis, McL. C. Wilson, G. E. Woodford, L. S. Ylvisaker.

There were also present Drs. S. R. Benedict, N. R. Blatherwick, T. G. Dabney, J. M. T. Finney, Otto Folin, Yandel Henderson, E. J. Ill, A. R. Rose, G. L. Schadt, S. C. Stanton, P. V. Wells and Messrs. J. K. Gore, W. M. Strong and J. S. Thompson.

Total attendance at all sessions, 152.

On motion the roll call was waived and the members were requested to register their names in the book provided for that purpose.

The names of the following candidates recommended by the Executive Council for membership in the Association were presented:

Dr. Hiram H. Amiral, State Mutual Life Assurance Company, Worcester, Mass.

Election of New Members

3

- Dr. Charles C. Berwick, Metropolitan Life Insurance Company, New York City.
- Dr. William Bolt, New York Life Insurance Company, New York City.
- Dr. Frederick R. Brown, New England Mutual Life Insurance Company, Boston, Mass.
- Dr. Edward J. Campbell, New York Life Insurance Company, New York City.
- Dr. Hugh W. Crawford, Connecticut General Life Insurance Company, Hartford, Conn.
- Dr. Francis C. Evers, New York Life Insurance Company, New York City.
- Dr. William D. Heaton, New York Life Insurance Company, New York City.
- Dr. Frank W. Kenney, Capitol Life Insurance Company, Denver, Colorado.
- Dr. Francis A. McGreen, New York Life Insurance Company, New York City.
- Dr. Roscoe W. Pratt, New York Life Insurance Company, New York City.
- Dr. William W. Quinlan, Mutual Life Insurance Company, New York City.
- Dr. William B. Smith, Connecticut Mutual Life Insurance Company, Hartford, Conn.
- Dr. Earl V. Sweet, Mutual Benefit Life Insurance Company, Newark, N. J.
- Dr. William S. Timblin, National Life Insurance Company of U. S. A., Chicago, Ill.
- Dr. Wallace Troup, Metropolitan Life Insurance Company, New York City.
- Dr. A. Burton Wilkes, Metropolitan Life Insurance Company, New York City.
- Dr. Edmund W. Wilson, Metropolitan Life Insurance Company, New York City.
- Dr. Arthur W. Young, Sun Life Assurance Company of Canada, Montreal, Canada.

On motion made by Dr. Huston and duly seconded, the Secretary was instructed to cast a ballot in favor of the election of each of these candidates.

The Secretary announced the ballot so cast, and the candidates were declared elected to membership in the Association. The President appointed Drs. Whitney and Pollard a committee to introduce the newly elected members to the Association.

It was announced that the following persons were present at the meeting as delegates from the American Life Convention:

Dr. B. F. Byrd, National Life and Accident Insurance Company, Nashville, Tenn.

Dr. J. L. Crawford, Cedar Rapids Life Insurance Company, Cedar Rapids, Iowa.

Dr. J. D. Heacock, Protective Life Insurance Company, Birmingham, Ala.

Dr. C. A. Heiken, Home Life Insurance Company, Philadelphia, Pa.

Dr. J. E. Kinney, Farmers Life Insurance Company, Denver, Colo.

Dr. W. T. McNaughton, Old Line Life Insurance Company, Milwaukee, Wis.

Dr. E. L. Mathias, Midland Life Insurance Company, Kansas City, Mo.

Dr. H. L. Metcalf, Springfield Life Insurance Company, Springfield, Ill.

DR. PATTON—We are very glad indeed to have these delegates with us.

On motion the reading of the minutes of the Thirty-ninth Annual Meeting of the Association held on October 25 and 26, 1928, was waived.

The Secretary read the minutes of the meetings of the Executive Council of May 16, 1929 and October 23, 1929. On motion these minutes were adopted as read.

DR. PATTON—It is customary at our meeting to have some comments made regarding those members of our Association

who have passed from us during the year. Because of the time that has been consumed, it has been deemed advisable the last few years to simply have the names read by the Secretary with leave to print the comments made by the representatives of the companies involved concerning the obituary notice. The Secretary will now read that list.

DR. BROWN—It is with deep regret that we announce that three members of our Association have died during the past year:

Dr. Frank W. Chapin, Medical Director, Home Life Insurance Company, New York City.

Dr. Henry A. Martelle, Assistant Medical Director, Connecticut Mutual Life Insurance Company, Hartford, Conn.

Dr. Harry Toulmin, Vice-President and Medical Director, Penn Mutual Life Insurance Company, Philadelphia, Pa.

The following memorials have been prepared:

FRANK W. CHAPIN, M. D.

(Presented by Dr. Chester F. S. Whitney)

Dr. Frank Woodruff Chapin died at his home, Wilton, Connecticut, October 9th, 1929. He was born at Erie, Pennsylvania, March 3rd, 1854, the son of Marvin and Marion Orinda (Cumins) Chapin. He graduated from Harvard University in 1876. After his graduation he entered Bellevue Hospital Medical College, graduating in 1879. He was appointed Assistant Physician at Riverside Hospital, N. Y. in 1880 and was made Resident Physician in 1882. He entered this Hospital at the suggestion of Dr. Edward G. Janeway, with whom he was associated for ten years, in order to take advantage of the little flurry of small-pox prevalent at that time and while on duty at the Hospital the epidemic of typhus fever broke out. In 1882 he resigned this post and was appointed to a Sanitary Inspectorship in the New York City Department of Health. In 1883-84 he went abroad for travel and study and was afterwards Diagnostician or Inspector of Contagious Diseases in the New York City Health Department. In 1886-89 he was Assistant to the

Chair of Practice at Bellevue Hospital Medical College. In 1888 he was Physician to the Out-Patient Department of the Presbyterian Hospital. He practiced in Astoria, Long Island for a year.

He was Associate Medical Director of the United States Life Insurance Company for two years, leaving that post in 1892 to become the Medical Director of the Home Life Insurance Company of New York, which position he held at the time of his death.

He was a member of the Harvard Club of New York, Harvard Club of New Jersey, Academy of Medicine, New York County Medical Society, Manhattan Medical and Surgical Society, Association of Life Insurance Medical Directors of the United States and Canada, Laurentian Club and at one time was President of the Alumni Association of Bellevue Hospital Medical College.

His widow, Mrs. Roberts Mott Chapin and two daughters, Mrs. Richard Pegram Myers and Mrs. Wayne Marshall survive him.

Dr. Chapin was a modest, conscientious and sincere physician with a rare fund of quiet humor. His memory will always be held in affectionate remembrance by his friends and co-workers.

HENRY A. MARTELLE, M. D.

(Presented by Dr. Charles D. Alton)

A span of fifty years is far too short a life, especially when a man has found the calling of his choice and has already shown efficiency in his work. Dr. Martelle came to the medical staff of the Connecticut Mutual Life Insurance Company in 1915, having already had some experience in the medical department of The Travelers. In his new position as Assistant Medical Director he devoted himself with commendable zeal to the new problems confronting the Company; he was especially helpful in the consideration of sub-standard business. The development of the Company's new laboratory received his assiduous, almost

Memorial of Dr. Martelle and Dr. Toulmin 7

affectionate, care and his visits to study the chemical equipment and processes of other companies resulted in expressions of friendship from many medical officers. His was a happy faculty of pleasantly impressing people and as with medical directorates so with examiners in the field and the Company's agents. His warm hand shake and cheery greeting gave an added value to his services.

Dr. Martelle was born at Richmond, Maine, on August 8th, 1879, and, following a brief attendance at Bowdoin College, he matriculated at Johns Hopkins University, from which he graduated in 1905. An internship at the Hartford Hospital still further equipped him for his professional work and in all these educational steps he acquitted himself with credit.

During the World War he served as a lieutenant in the Medical Corps at Camp Mead, Maryland.

His death came as a sudden blow to his friends and it was only afterward that we realized how in the midst of his activity and devotion to duty he had been carrying the consciousness of a malady which was sooner or later to prove fatal.

His associates are deeply conscious of a grievous loss and will long retain the memory of duty well performed, remembering that the good men do lives after them.

HARRY TOULMIN, M. D.

(Presented by Dr. James P. Hutchinson)

Dr. Harry Toulmin, Vice-President and Medical Director of the Penn Mutual Life Insurance Company, born November 4, 1865; member of the Association of Life Insurance Medical Directors; died February 8, 1929, of carcinoma of the rectum, after an illness of two years.

Dr. Toulmin was educated at Antioch College, and entered Lehigh University in the Fall of 1882, receiving the degree of Ph. B. in 1886. He entered the Medical School of the University of Pennsylvania this same year, where he was a member of the Phi Beta Kappa, and graduated in 1889, receiving the

degree of M. D. He was interne under Sir William Osler at Johns-Hopkins Hospital, from May, 1889, to September, 1890, following which he practiced medicine in Baltimore, from December, 1890, to January, 1892. He then moved to Philadelphia, where he took up the practice of medicine and became Instructor of Physical Diagnosis at the University of Pennsylvania.

Dr. Toulmin was Home Office examiner of the Penn Mutual Life Insurance Company, 1892-1898. In the latter year he was elected Assistant Medical Director of the Northwestern Mutual Life Insurance Company, Milwaukee, Wisconsin, and acted in this capacity from 1899-1900. He was then elected Assistant Medical director of The Penn Mutual Life Insurance Company, and returned to Philadelphia, serving as Assistant Medical Director from 1900-1910. He was elected Medical Director in that year, and in January, 1923, was elected Vice-President and Medical Director, serving in that capacity until his death.

Dr. Toulmin was a Fellow of the College of Physicians at Philadelphia; a member of the Association of Life Insurance Medical Directors, and of the American Medical Association. He was Vice-President of the Philadelphia Health Council and Tuberculosis Commission. He was Vice-President of the Association of Life Insurance Medical Directors from 1910-1912, and President from 1912-1913. During the World War he was in charge of First Aid for Southeastern Pennsylvania Red Cross.

Dr. Toulmin took a keen and lively interest in the affairs of the Medical Directors Association at all times, and at the time of his death was a member of the Executive Council.

During his year's illness Dr. Toulmin maintained a cheerfulness and equanimity that was courageous. His death was a loss to The Penn Mutual Life Insurance Company.

DR. PATTON—Next in order is the report of the Nominating Committee.

The Secretary read the following report:

"The Nominating Committee of the Association of Life Insurance Medical Directors begs leave to report that at a meeting

Report of Nominating Committee

9

regularly held on October 23, 1929, the following nominations were suggested:

For President—Dr. William Muhlberg,
For First Vice-President—Dr. Robert L. Rowley,
For Second Vice-President—Dr. Charles L. Christiernin,
For Secretary—Dr. Chester T. Brown,
For Treasurer—Dr. Albert O. Jimenis,
For Editor of the Proceedings—Dr. Robert A. Fraser,
For Members of the Executive Council—Dr. George A. Van Wagenen, Dr. Edwin W. Dwight, Dr. Morton Snow, Dr. Eugene F. Russell, Dr. Ross Huston.

All of which is respectfully submitted.

ROBERT M. DALEY,
CHESTER T. BROWN,
ANGIER B. HOBBS,
WILLIAM R. WARD,
CHESTER F. S. WHITNEY,
THOMAS H. WILLARD, *Chairman.*"

DR. PATTON—You have heard the report of the Committee. The officers' election will be tomorrow morning and if there are no other nominations, motion to close the nominations and instruct the Secretary to cast the ballot for the list as nominated by the Committee will be in order.

No further nominations being presented, it was moved, seconded and carried that the nominations be closed, and that the Secretary be instructed to cast the vote for the ticket as nominated.

The Treasurer, Dr. C. L. Christiernin, read his report. The Auditing Committee, Dr. Whitney and Dr. Huston, reported that the Committee had audited the Treasurer's accounts and found them to be correct. On motion made and seconded, it was ordered that the report be accepted and placed on file.

DR. PATTON—We now reach the reports of our Committees and I see Dr. Rogers is with us. I will be very glad to have his report of the M. I. B. Committee.

Dr. Rogers, Chairman of the Special Committee in charge of

the M. I. B., presented the report of that Committee, which was accepted with thanks and ordered placed on file.

The Secretary announced that the following amendment to the By-Laws had been recommended by the Executive Council:

That hereafter delegates from the American Life Convention or other groups who attend the annual meeting of the Association be relieved from paying dues as prescribed in Article 1, Section B, of the By-Laws.

On motion duly made and seconded, the amendment was adopted.

DR. PATTON—I am very glad indeed to have the Association meet with us today and we will have a word brought to us by Mr. John K. Gore, Vice-President and Actuary of the Prudential.

MR. GORE—Mr. President and Gentlemen. Mr. Duffield has asked me to express his deep regret in not being able to welcome you in person this morning. He is attending a meeting of the Trustees of Princeton University and as that Board meets only four times a year he felt himself obliged to be present. Mr. Duffield and all the other officers of the Prudential extend to you a most cordial welcome. We are glad that the Association of Life Insurance Medical Directors is meeting here, because we realize keenly the important work that has been and is being accomplished for the institution of life insurance by your organization.

As far as a mere layman can judge, you have ahead of you an especially interesting and constructive program, and I congratulate you that among those who are to take part in your proceedings, in addition to your own members, are such distinguished men as Dr. J. M. T. Finney, Sr. of Baltimore, Dr. Edward J. Ill of Newark, and Dr. Yandell Henderson of Yale University. There seems to be a good deal of Prudential on the program but it is not for me to find fault with that. I think it shows that the Prudential boys are standing by their chief, the Presiding Officer.

The tremendous industrial expansion in the United States and Canada in the last decade and the corresponding developments in the business of life insurance have greatly emphasized our problems of selection, especially in the consideration of applications for large amounts of life insurance and in connection with insurance giving total and permanent disability. These problems are only too familiar to all of us. A great many of you were present at the joint meeting of your Association and the Actuarial Society last May, when this subject was discussed and a number of valuable suggestions offered. Gentlemen, it is to be hoped that you and the actuaries together will soon develop working plans for meeting these difficulties successfully.

The morning papers brought us the sad news of the death of Mr. Louis Butler, President of the Travelers Insurance Company. A great many of us knew Mr. Butler well and I am sure we all sympathize with his family and his fellow workers.

Your time is too valuable for me to take any more of it. We want you all to feel very much at home, gentlemen, and I trust your meetings will be both profitable and enjoyable.

DR. PATTON—I want to add to what Mr. Gore has had to say that we have made every effort we could to take care of your wants and needs. If you find we are not doing so, please let us know and we will try to take care of the want or need for which we have not provided.

Usually in the presence of this body, there are a few words—or address—or whatever you want to call it, from the President of the Association. What I have to say will be very brief as I hope the material as prepared for the program is going to fill our time. I have taken the topic of Life Insurance Selection.

LIFE INSURANCE SELECTION.

J. ALLEN PATTON, M. D.

Medical Director, The Prudential Insurance Company of America.

The decision as to the insurability of an applicant for life insurance, and the kind of policy that can be issued, rests for

the most part with the Medical Department—aided and abetted by the Actuarial.

This has become very well recognized, even in these modern days, when many companies are writing so much non-medical business.

Life insurance medicine is primarily the ultimate prognosis or the final result of treatment. It is based upon the history as given to or obtained by the examiner, together with his physical examination records and his opinion of the applicant at the time, and in addition upon the experience of the companies with insured groups of similar individuals. Modern methods of examination, diagnosis and treatment with clinical prognosis, must be known and considered in weighing an applicant for insurance.

The costs to the company are estimated from its experience with insured individuals. These naturally fall into groups based upon physique, personal and family histories, occupation, etc., and the examination history and findings of present or previous diseases.

Studies of our past experiences with kinds and amounts of policies—with standard and substandard individuals and groups—occupations—accidents and disabilities—physical impairments, etc.—have led to quite definite classifications by the Medical and Actuarial Departments. Instructions based upon such studies have been formulated for the use of the field and the Home Office forces. The watchful supervision by the two Home Office Departments that are responsible for these instructions is necessary to maintain a proper mortality experience.

Based upon present day knowledge, I believe that a company's average or usual mortality can be maintained if the examination and other reports are first carefully reviewed by the laymen trained for that purpose in the Home Office. These laymen, according to the rules that have been established for action, either approve the application or refer the papers to the Medical. Applicants that are all right in all respects or that come definitely within the rules outlined for their instruction are readily considered by lay approvers or reviewers. The medical directors

receive those cases that can not be classified as above. Thus, in our office, 80 to 85% of the applications in number are handled without direct or immediate review by or consultation of the medical directors. Maintenance of the proper desired mortality requires constant study of the changing conditions that our population experience.

Instructions for both the Home Office and Field must be continually revised and brought up to date. This requires frequent conferences of those responsible at the Home Office and also direct contact by them with the field men, so that these men can be given at first hand the general reasons for the actions taken. I am sure that the establishment of personal acquaintanceship between the field men and the Home Office selection departments will make for less complaints and bring about a more general understanding by all concerned.

Though medicine is called both an Art and a Science, there are many persons who think it is neither when applied to life insurance selection. Science is defined as knowledge and Art as the application of knowledge to practice. I have never been able to agree with the statement made by the executive of one of our companies that "the Medical Department was the only part of a life insurance company that was not scientific". All sciences are not exact and we frankly admit that medicine is not. The art in life insurance medicine exists in applying definite medical facts and conclusions to life insurance so that a desired mortality is obtained. Certainly definite progress is made each year towards a more scientific state in medicine and I firmly believe that life insurance medicine is maintaining its position in this respect.

No exact science has as intricate a structure to study as the human body, composed of bone and flesh, blood and nerves, a central brain and heart, a digestive and urinary system, a process of metabolism and nutrition, all so intimately inter-related and under the control of the mind. Is it any wonder that it has for all time been the seat of intense study for the psychologist, the physiologist, the pathologist, the doctor and the surgeon. All these must be united in the Medical Director.

The Accidental Death Benefit and Disability Income features have materially added to the problem the last few years. These foster children must be put in their rightful places in the family so that amity and accord may exist. There is a distinct difference in medical and insurance selection, expectation and experience, between life and disability insurance. Companies must get their proportionate share of the business and they must not have too much indigestion in assimilating these provisions that are being added to their life exposures.

One of the hardest problems we have to meet is the complaint from the field force, often brought to our attention through appeal to the executive, that their applicant has been given better consideration by some other company with a well established reputation for strict selection: viz., a better rating, approved while we rejected, or given Disability Income or Accidental Death Benefit, etc., when we have not.

Uniformity in action, or at least greater uniformity, is certainly a desirable goal for us to attempt to reach.

There has been considerable progress towards this millenium during the last ten years and one sees signs of greater efforts in that direction, with selection of risks becoming more uniform year by year.

The M/A reports of 1912 were a great step forward and these have been supplemented by individual and joint studies reported from time to time at our meetings, and those of the actuarial organizations; by the joint occupational study that has just been completed; and by the new M/A work that has just gotten well under way. Thus we have been furnished the material for group selection. Whether individual selection can ever be expected or followed is for the future to determine.

The adoption of the Numerical basis by a number of companies for their substandard business has meant much for uniformity, and we all owe our thanks to the New York Life and its representatives for their pioneer work.

During this last year, the Disability Income question and the applications for large amounts became so prominent that an

informal joint meeting was held by the Actuarial Society of America and our Association for the discussion of these features. A committee was appointed from these two bodies by their presidents, with the approval of their executive councils.

This committee held an organization meeting and after considerable discussion some recommendations were prepared for its further consideration.

This committee will get together again in the near future and will report the results of their work to the parent societies.

The executive must be consulted, for the general business policy of the companies is encountered. That is outside our work and it is not our duty to reason why along that line. The Actuarial and Medical Departments can determine the lines and limits of safety and good selection. They can state the reasons for caution and uniformity. The final decision is executive.

The combined opinions and recommendations of our two organizations after thorough consideration by them must affect materially the final action that will be followed. The proper interchange of information for the protection of the business in general is a necessity.

The question of a uniform blank has from time to time come up for discussion and while there is some union on particular questions yet there will always be individual ideas as to the relative importance of certain matters, and these ideas—when joined with a company's experience—will always lead to slight differences in the blanks used. The general lines of selection are not affected to any extent.

One problem that will make for greater uniformity belongs to us and is now being given consideration by a committee of this association—a method of selection and appointment of qualified examiners. Considerable time and thought has been given to this problem and we hope that we are nearer the solution. Life insurance medicine must be given its place in our medical schools and the various medical, surgical and special societies must recognize that this is a specialty just as much as is eye, ear, chest, obstetrics, gynecology, etc.

Better examinations will be made when the medical profession is brought to a realization of the above. Accurate and complete health histories with the results of physical examinations by a doctor of average ability will produce better results than inaccurate or incomplete or careless histories or examinations by a highly trained one.

Borderline cases will always be with us, for we must continue group selection in the main and can go to individual selection very rarely. Cases that are near the line always appear to the interested applicant and agent as being on the favorable side of the line and entitled to approval instead of rating or rejection or to a more favorable rating than the one offered by the Medical Department.

Confusion will arise in any Home Office if the selection limits are broken or let down for any case. Be sure of your information and of your lines of reasoning and then adhere to your decision that you feel is right to take care of the physical hazards in the case under consideration.

One might continue this indefinitely but I do not intend to discuss special groups or cases. I do feel that we are progressing towards a more common consideration of our applicants and I sincerely trust that this meeting of our Association will see us farther along on our way.

* * * * *

Means and methods used to test the efficiency of the circulatory system, to detect abnormalities when present and to measure the degree of change from the normal, are all of value to the medical director.

A study of these procedures and of a group of cases selected following their use will enable us to go a long way towards determining their value.

Oscillometry, as practiced and used in France, has received limited attention in America and I agree with Dr. Brathwaite that now is a good time to bring this method to the notice of our Association.

Brathwaite—Cardiography and Oscillometry 17

DR. BRATHWAITE—Mr. President and Gentlemen: I have really nothing much to add in connection with this paper, which is, as you perfectly well see, not original. It is merely a plea, so to speak, for the study by us as Medical Directors of life insurance companies of the use of instruments of precision when they appeal to us as being of assistance in our work.

The first part of this little paper is simply a plea for a more extensive use of the electrocardiograph. It isn't necessary for any of us, I think, to emphasize the importance of this. If we will only really make use of the opportunities which are presented to us by these instruments of precision, we will improve our selection.

The second part of the paper refers to a subject a little less generally known; I refer to the art of oscillometry. It is well known abroad, but not so well known in the United States. I don't know why. I fancy because the literature is practically entirely in the French language. There is practically nothing in the English language in reference to it, German and French being the languages of the bibliography. The oscillometer was used many years ago and a description of it is contained in this paper and the use of it as described by Professor Pachon, Professor of Medicine in the University of Bordeaux.

Oscillometry is generally used in France. Vaquez has used it for years. Some of the hospitals in Paris use it as a regular part of their diagnostic equipment. It is very simple; the physics of it is quite easy to understand. It has, we think, value from our standpoint as described. I have an oscillometer here which I shall be glad to show to anybody who would like to see it.

I would like to show for a few minutes only on the screen some slides illustrative of electrocardiography, which have been generously loaned to us by Dr. S. Calvin Smith of Philadelphia, whose reputation and whose book you all know so well. Following that I have four slides which we made ourselves being a combined electrocardiograph and oscillometric record, together with a manometric index, on one film, which has never been done before, and perhaps it may be of some little interest to

you. As I am by no means an adept in the interpretation of the electrocardiograph, I am going to ask my friend, Dr. Ungerleider, who is in charge of the Equitable's diagnostic laboratory, to explain them to you and then I will describe the last ones, which combine an oscillometric and electrocardiographic record, with the hope that you will be enough interested to pursue the subject and ascertain whether or not in the future our investigations will lead us to the belief that we have a valuable aid in our work.

DR. UNGERLEIDER—These slides are rather primary slides and they all explain themselves.

Dr. Ungerleider presented a number of slides with an occasional comment.

DR. BRATHWAITE—I would like to explain the last four a little more in detail and would ask you to look at some of these oscillometric observations. These are somewhat different and a little new; in fact, that particular film from which we made that glass slide was only done the day before yesterday. Below is the manometric index. In other words, the height of that line on the film represents the pressure in the cuff when the oscilometer is applied, and from the paper, if you have read it, you will see that one begins an oscillometric observation by producing a pressure of 200 mm. of mercury in the cuff and reduces it by 10 or 20 mms. going on down. This line shows the manometric pressure. In other words, you go from the bottom up. This one, we will say, is 200 mms. of mercury in the cuff and this one up there is 180. They are going down by 20 mms. Consequently the distance between the margin of the film and the index here is less and, as you will see in two or three more, it decreases, showing one exactly what the pressure is, because it is measured on the slide which is divided into partitions, which you see. The oscillometric index is the next one, and that one is at a high pressure in the cuff of 190 or 200 mms. of mercury. The result is that you see the oscillation is very slight, which is explained to you in the paper, that is, the reason why such is the case. Above we carried along at the same time on the same film the ordinary electrocardiographic record, so that we have on

Brathwaite—Cardiography and Oscillometry 19

the same film simultaneously an opportunity of studying the function of the heart by the electrocardiograph. We study the resiliency of the vessel by the oscillometer and the manometric index gives us on the same film the amount of pressure which we are using in the cuff.

Next slide. You will see here that as we decrease the pressure in the cuff the oscillations become wider and that the maximum oscillation of each wave is in diastole and the observation thus becomes a study of diastolic pressure. This has been done for the purpose of this demonstration whereas an oscillometric observation may be obtained by use of the oscillometer itself manually and by observing the needle without going to the trouble of transferring its amplitude to a film as we have done here in this instance. Above the oscillometric tracing in each of these slides is the electrocardiographic record going along simultaneously so that you have objectively the two records to look at as the phenomena are thus graphically depicted.

Next slide. As we decrease the pressure, the oscillations increase, you will notice, and this is a particularly important thing inasmuch as it represents a study of the diastolic pressure.

Last slide. Here you will notice the amplitude of oscillations are fading away due to the decreasing pressure in the cuff under that of the diastolic and if we carried it further still we would get a perfectly flat line with no oscillations from which point we started.

CARDIOGRAPHY AND OSCILLOMETRY IN LIFE INSURANCE SELECTION.

FREDERICK G. BRATHWAITE, M. D.

Associate Medical Director, Equitable Life Assurance Society.

In physical diagnosis it is becoming more and more a standard practice for diagnosticians to avail themselves of instruments designed for recording precisely certain phenomena which otherwise would escape accurate record under the older, and to a great extent, obsolete methods of rely-

ing upon the tactile, aural or other senses.

Life insurance practice has been proverbially slow in following clinical advances.

Latterly, however, owing to the apparent increasing importance of cardio-vascular mortality, the use of instruments of precision is at least desirable, if not mandatory.

This paper is intended to show that the use of the electrocardiograph (cardiography), and oscillometer (oscillometry) are essential for a thorough study of cardio-vascular conditions. We will not dwell at this time on radiography and fluoroscopy, also valuable adjuncts.

CARDIOGRAPHY.

A well known authority (*) has stated that persons who have conventional signs of heart disease, being aware of physical findings, do not as a rule expose themselves to the stigma of rejection, arguing that life companies are not generally subjected to the necessity of determining the fitness of applicants with such signs as shortness of breath, limitations on effort, murmurs, enlargement, etc. This contention, especially in late years, must be modified, especially by reason of the development of substandard business, in which just such applicants *do* present themselves and may not be perfectly frank in their description of symptoms of possible myocardial disease.

It will thus be seen that there is stronger argument for cardiographic records than was at that time fully appreciated.

The argument for graphic heart records was limited in this paper to those whose average age is young, possibly 30 years, of whom the writer said, "Therefore, if the heart has some defect as yet unrevealed * * * the defect is usually not manifest in physical signs".

Further it was said that since such masked possibilities may invalidate the customary methods of heart appraisal, it

*S. Calvin Smith, Journal of A.M.A., April 7, 1923, "The Life Insurance Value of Graphic Heart Records".

Brathwaite—Cardiography and Oscillometry 21

is of definite value to know that in such apparently healthy men the recently introduced methods of cardiography examinations often yield evidence of unsuspected heart conditions which, were they to continue until the age of perhaps 45, would have, by that time, so progressed as to be manifest in physical signs of heart fault sufficiently pronounced to constitute cause for rejection of the applicant.

On the other side of the picture he alludes, however, to certain irregularities accompanied by auscultatory phenomena which cause the cautious examiner to criticize the applicant as a potential heart risk even though the life histories of persons similarly affected have repeatedly shown the error of such judgment; thus demonstrating that had a heart record been made at the time of the examination it would have been possible to have identified the irregularity as of little or no pathological importance, warranting acceptance if the selective process had been fortified by a physiological record written by the heart itself.

And again "It is perfectly possible for the more severe focal infections and toxins to produce clinical symptoms of heart muscle inefficiency to a degree that simulates actual structural heart disease with the attendant physical signs of heart enlargement and abnormal signs. These symptoms are present as long as the toxic causes are present, but on removal of the cause the heart no longer protests, the symptoms abate, the physical signs disappear, and the heart is amply able to support the individual indefinitely".

The author of this paper expressed the opinion that insurance companies suffer an annual loss in premiums which in the aggregate must be enormous. Both sources of economic losses could have been avoided in many instances by the employment of cardiographic investigations.

Furthermore, in a personal communication with Dr. Smith he asserts "recent experiences conclusively prove that the electrocardiograph will reveal the impending possibility of coronary artery occlusion as much as two or three years

before clinical manifestations are present; and long before the patient presents any of the classical symptoms of the condition".

Without going into explanations involving highly technical considerations, we may summarize fairly the reasons for the use of the electrocardiograph in selection for life insurance, especially where large amounts are involved, where the applicant is of advanced age, or both, and where there is a reasonable presumption as to the existence of cardiovascular abnormality, as follows:

1. The determination of the presence or absence of coronary disease. This would permit of the acceptance of certain applicants who would otherwise be rejected and the rejection of applicants who would otherwise be considered acceptable.
2. The recognition of benign types of arrhythmia which under ordinary examination might suggest serious heart impairment; i. e., certain types of premature contractions or sinus arrhythmia.
3. Recognition of certain forms of myocardial affection.
4. The possibility of including in the record of applicants graphs written by the heart itself, rather than the interpretation of cardiac conditions by use of the tactile senses.

All of the above may be summed up in saying that we determine by cardiography the innocent nature of certain cardiac irregularities, have a graphic record made by the heart itself and positive evidence of structural impairment of the heart muscle impossible by the ordinary methods of examination now in general use. "In other words, the electrocardiograph will differentiate between those heart disturbances which are alleged to be 'functional' and those heart disturbances which are due to 'organic' (actual structural) heart impairment." (Dr. Smith.)

[As the electrocardiograph is in such general use (clinically) it is not germane to the subject that any description of this instrument of precision should be included here.]

Brathwaite—Cardiography and Oscillometry 23

OSCILLOMETRY.

Oscillometry is the measure of the pulse wave against resistance, as vascular resistance. Arterial blood pressure is constantly varying, that is, oscillating between maximum and minimum points. There is no fixed degree of arterial pressure. In oscillometry, we record the maximum and minimum points between which arterial blood pressure varies; the amplitude of oscillation. A tracing showing the variation of oscillatory amplitude at different pressures is called the oscillometric curve.

As the minimal pressure is that which is constantly borne by the arteries, it is evident, as demonstrated by Professor Pachon *, that this pressure must be considered before all others inasmuch as the maximal pressure represents only an *intermittent burden* to the vessels. But another reason other than that causing us to study the minimal pressure as one acting constantly against the vessels, is from the standpoint of the heart itself, inasmuch as the minimal pressure is that which is exerted against the aortic valve in ventricular diastole. It is, therefore, the *minimal pressure* against which the heart has to act at the moment of ventricular systole. Thus it follows that the ventricle in order to open the aortic valve must adapt its effort, in doing so, to the resistance which had caused it to close.

A normal minimal pressure requires an ordinary cardiac effort, and obviously, therefore, the effort must increase in proportion to any rise in minimal pressure. Whereas a high maximal pressure does not necessarily indicate a pathological condition, a high minimal pressure on the contrary represents *real hypertension* as it imposes on the heart an extra effort in systole. The efficiency of the heart may be studied by oscillometry and it is claimed that more is discoverable by this process than by that of clinical sphygmomanometry.

Professor Pachon has shown quite clearly that the extinction of the pulse below the compressed region in the original Riva Rocci experiment (compression of the arm with the

*"La Mesure de la Pression Arterielle par la Methode des Oscillations."

exploration of the radial pulse) was due, not as had been supposed, to an *arrest* of the flow of blood by arterial compression but to the *uniformization* of the flow of the blood itself in a very resilient and elastic segment, i.e., in an artificial aneurysmal pocket produced by the cuff when inflated as is shown by the fact that at the same moment that the radial pulse disappears, the compressed zone over the brachial registers appreciable pulsation. This is important as indicating the value of this method. When, as Professor Pachon expresses it, it was thought that the extinction of the pulse below the compressed zone was caused by arrest of the flow of blood it followed (on what was considered conclusive evidence) that the figure of the manometer read at the exact moment of such extinction represented the degree of maximum pressure. That was due, he said, by failure to understand that the arterial pulse (radial) was capable of extinction by another means, that is, *uniformization* as above described, due to the production of an artificial aneurysmal-like dilatation.

The first effect of brachial compression is to cause equilibrium by acting on the exterior surface of the humeral artery to one part of the pressure. The flexible pocket thus produced absorbs and deadens the pulse stream by its elasticity, the pulse disappears below by *uniformization* of the flow of blood which pursues its course beyond the flexible pocket continuously without pulsating; hence the disappearance of the pulse below the compressed point is the result of another cause than the arrest of the flow of blood by arterial obliteration, and the degree of the compression above has no further connection with the maximum arterial pressure.

This conception of the circulatory phenomena has the advantage of explaining simply two peculiarities known in the original method of Riva Rocci, first, the variation of the figures dependent upon the variation of the size of the humeral cuff; second, the weakness of the figures of maximum pressure obtained by this method in the normal person.

Brathwaite—Cardiography and Oscillometry 25

Finally as there is no comparison between the exploration of the pulse below and at the level of the compressed zone the exploration of the pulse below that zone represents an inaccurate criterion for a sphygmometric experiment. The exploration of the pulse at the same level of the compressed region only should be systematically followed in clinical sphygmomanometry.

The researches of Professor Pachon have thus determined the general law of sphygmometric exploration. The determination of this law as an immediate corollary has a peculiar consequence in that it imposes the method of oscillations as a method of choice and sphygmomanometry is therefore, modified in accordance.

THE SPHYGMOMETRIC OSCILLOMETER.

Professor Pachon has shown that the correct employment of Marey's experiment (in which it was demonstrated that when we increase the pressure from zero to say 200 mms. of mercury and subsequently progressively decompress, we observe pulsations in proportion to the degree of the fall of pressure thus produced) requires two fundamental conditions which in practice had not then been satisfactorily complied with. (1) A great sensibility; and above all (2) A constant maximum sensibility of the instrument indicating the pulsation. Great sensibility, in order to insure necessary clarity in the differentiation of the pulsation, and in order to allow one to understand easily the beginning and the end of the phase of the increase in oscillations which indicate in Marey's method the maximal and minimal pressure. Constant maximal sensibility is necessary for it is clear that without constancy of sensibility of a machine in dynamic operation in the different degrees of pressure under which it must function, all genuineness of comparison of the pulsation under these different systems disappears at once. Now the manometers and sphygmomanometers in ordinary use do not fulfill these requirements.

The conditions of the problem having been stated, Professor Pachon has proved its simple and complete solution with an entirely original machine, the sphygmometric oscillometer.

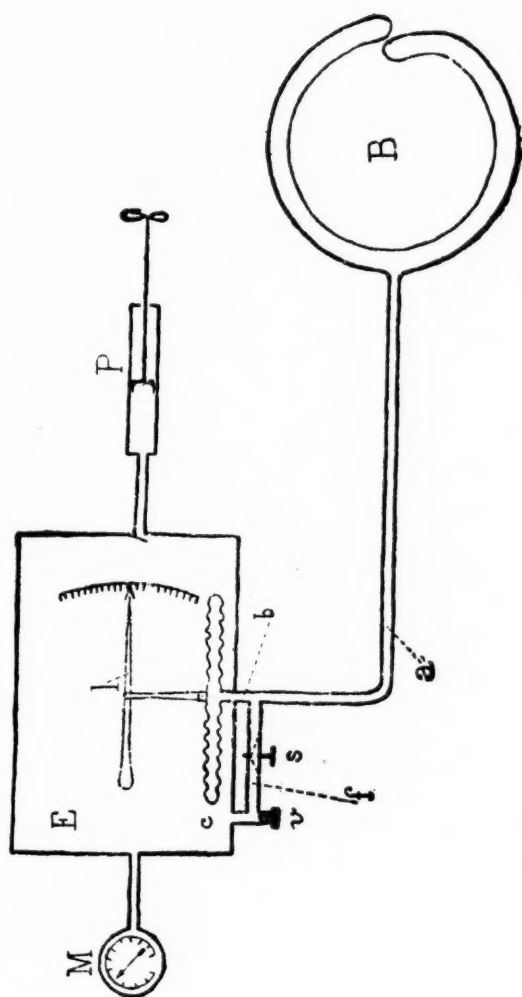
This machine constructed in accordance with new mechanical ideas constituted the first accomplishment of an elastic machine with a coefficient of resistance not only constant but to a great extent impervious to whatever type of pressure it might be required to interpret.

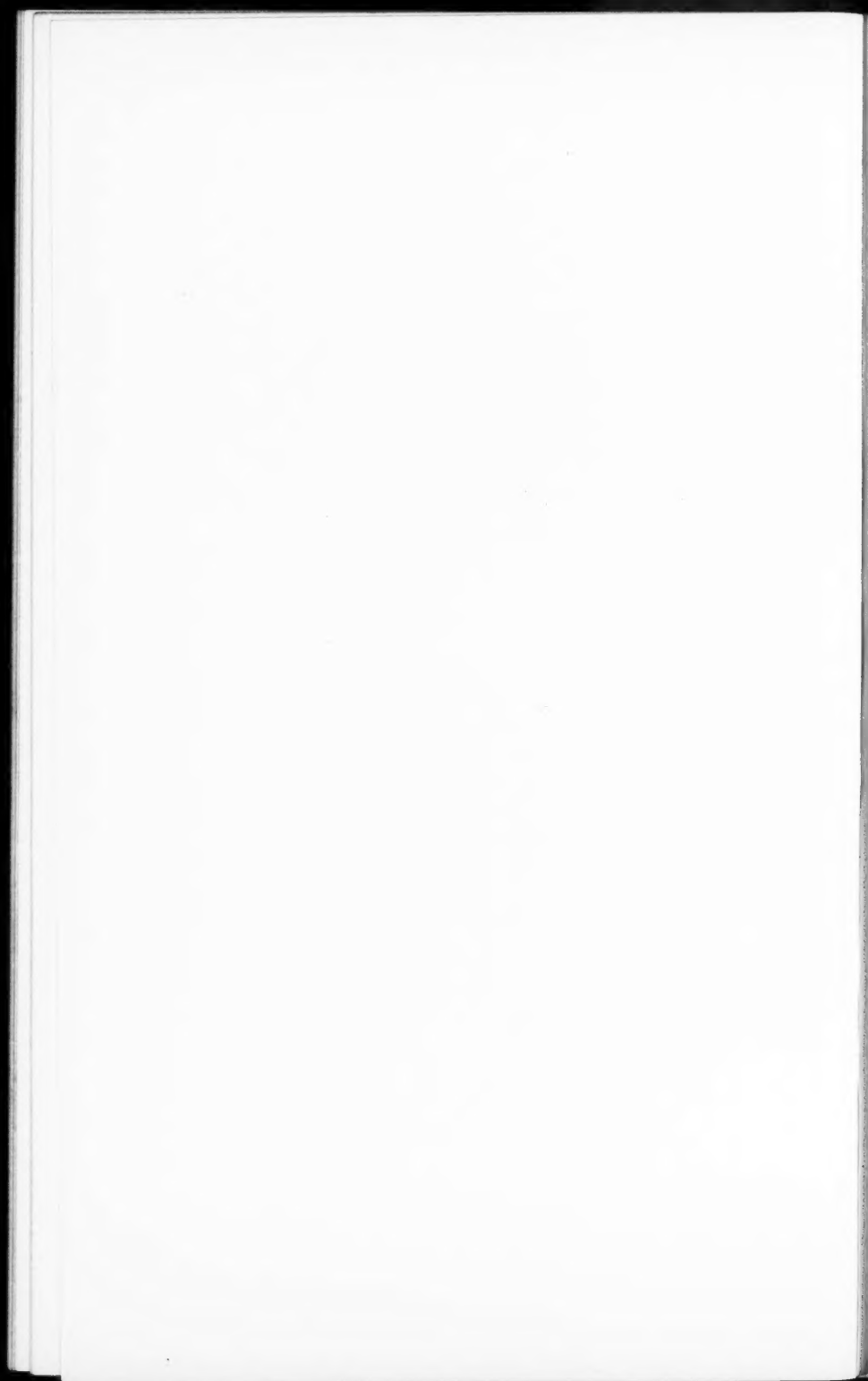
As the Oscillometer is not in such general use a brief description of its mechanical construction may be appropriate.

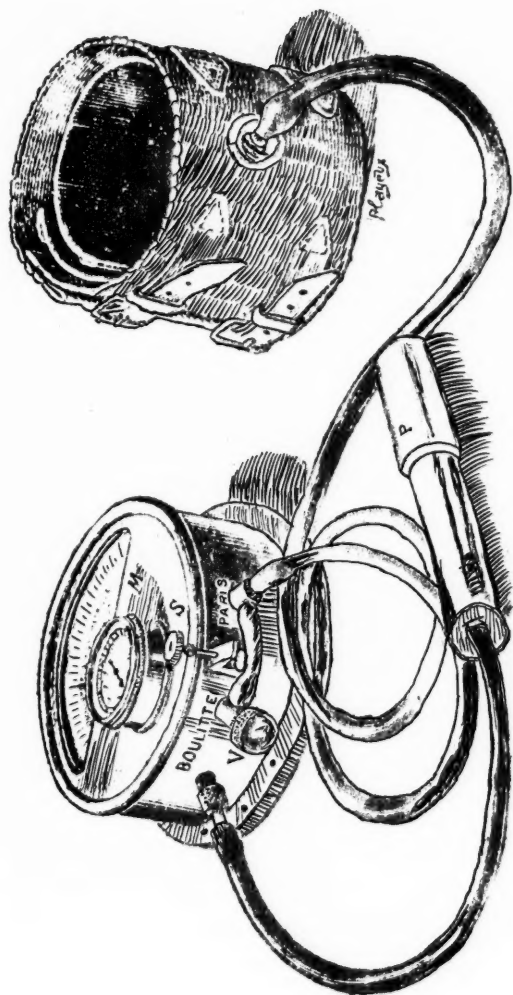
DIAGRAM OF THE SPHYGMOMETRIC OSCILLOMETER.

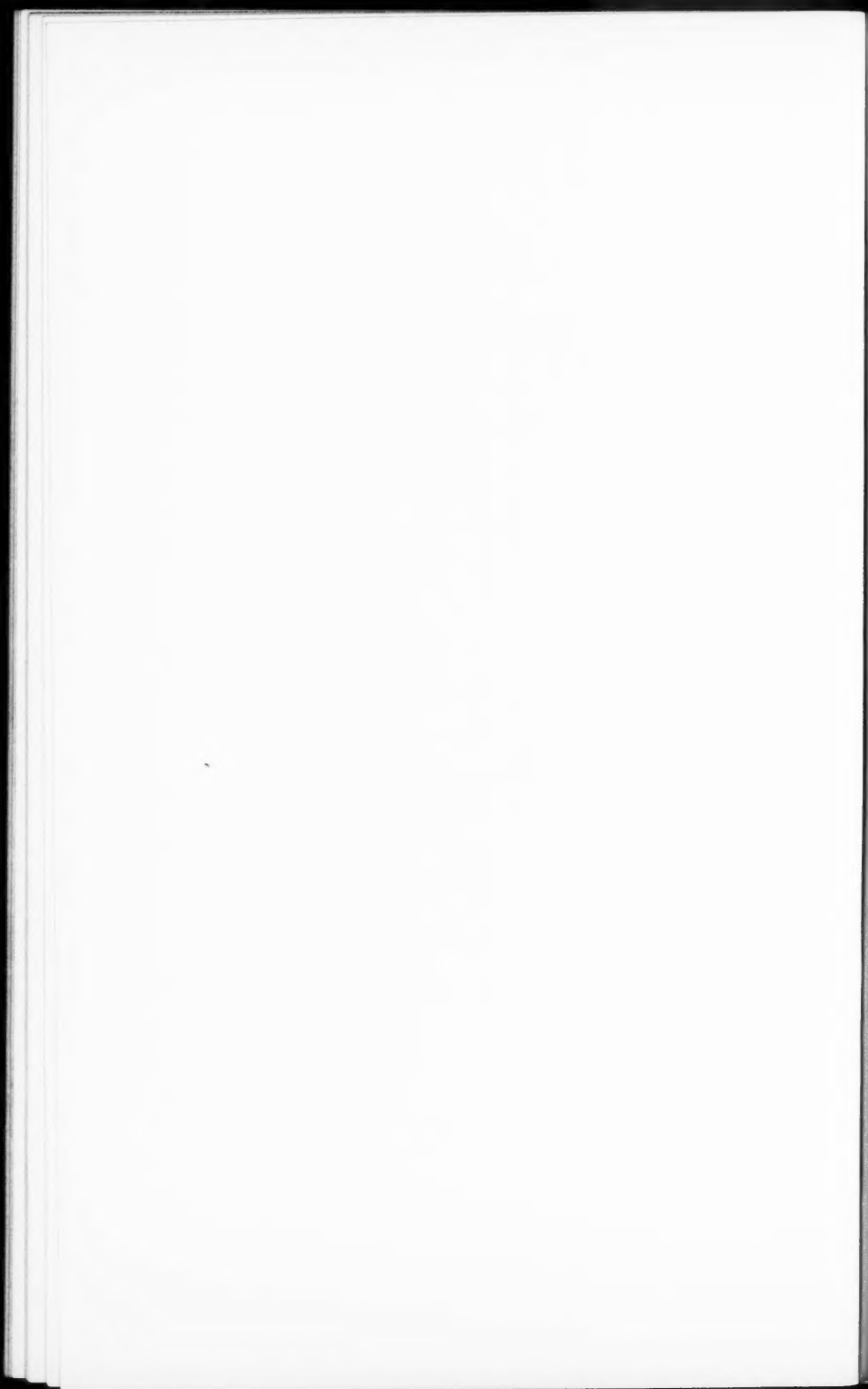
Within a rigid and hermetically closed metallic box E is placed an aneroid capsule C. B represents the cuff for application over the brachial artery. (There are other cuffs for other arteries.) The cuff B communicates with chamber E through two channels, b and f, and the aneroid chamber C (channel f.b.a.). The pump P forces air through the entire system to any desired pressure which is measured by the manometer M in mms. of mercury. The pressure so obtained can be reduced by the operator releasing the escape valve V. It is thus seen that the pressure within the capsule C is always equal to the pressure within the chamber E since C and E communicate through channel f. At no instant therefore, are the walls of the capsule (which constitutes the oscillometer proper) distended, whatever may be the degree of pressure inside the entire closed system. When the aneroid capsule C is in operation by use of the cut-off S, the registering needle 1 oscillates through an arc over a scale of *arbitrary* units from zero to 20. The pressure which has been produced by the pump P in operation may be made to register on the manometer M up to 300 mms. of mercury.

If the instrument be left alone the registering needle of the oscillometer remains motionless even if the pulsations are occurring in the cuff because these pulsations are transmitted both









Brathwaite—Cardiography and Oscillometry 27

to the internal surface of the aneroid capsule (via B) and to the external surface of the same (via the metallic box). Thus the state of equilibrium which exists on both sides of C renders the capsule immobile.

S is a cut-off (with digital pressure of the operator) by means of which the system c.b.a. can be completely shut off from the large chamber E.

The variations in pressure set up in the cuff by the rhythmical variations in the volume of the segment of the arm under compression are *now* transmitted exclusively to the aneroid capsule which registers them by means of the oscillating needle at all degrees of counter-pressure with *constant and maximal sensitiveness*, because the *tension* of its walls, no matter what the pressure may be, *is always nil* until the separator key is depressed. The oscillations of the needle thus represent the variations of the pulse wave.

The oscillometric curve as shown by the amplitude of the excursions of the needle is a measure of the cardiac impulse.

GENERAL VIEW OF THE SPHYGMOMETRIC OSCILLOMETER.

Given a series of pressures and the desirability of studying their significance, i.e., to analyze the amplitude of arterial pulsation, it is necessary to use a separate part of the mechanism S whose action intercepts the communication between the case E on the one part and the lead composed of the arm band B and the manometric capsule C. By compressing the stop-cock S the variations of pressure created in the cuff are transmitted exclusively to the manometric capsule with a constant and maximum sensibility. Thus it will be seen that besides its use as a sphygmomanometer it allows us to study, by reason of its great sensibility, arterial pulsations; and to understand cardiac force especially in connection with arrhythmias, and in the modern investigation of the heart's work. Indirectly it becomes of great use in the study of the elasticity of the vessels.

To explain this more fully we quote from an article by Dr. Georges Cuvier (Bordeaux) from the "Carnet Medical Français," April, 1922.

"Oscillometry is based on one fundamental tenet; the oscillometric curve. This 'maximal oscillation' observed during an 'oscillometric exploration' takes place when the pulse wave meets with the least vascular resistance.

"The wall of the artery, reacting at this moment, produces an oscillation which is exactly proportional to the value of the cardiac impulse. From this may be drawn the first law of interpretation: *Under equal conditions of arterial exploration the oscillometric curve gives the value of the cardiac force.* (Pachon.)

"The oscillometric curve is, further, the expression of the total pulse of the segment of the limb under experiment. It is caused by the partial expansions of the different arteries that give rise to it. It is controlled by the expansible arterial surface, that is, by the more or less important diameter of the artery. It becomes thus a direct element of knowledge of peripheral circulation, both from the point of view of vascular tone or vasomotor activity as well as from that of organic arterial resiliency. The second law of interpretation may be drawn from it: *Under equal conditions of cardiac impulse, the oscillometric curve gives the value of the vascular section, that is to say the state of contraction or expansion of the arteries explored.* (Pachon.)

"The variations of the oscillometric curve are consequently the function of two elements; the one, cardiac, the other, vascular, and these are only of specific value in the variations of one of the two, if the other is already known or remains constant during the exploration. Hence, the conclusions: The comparison of the respective values of the curve cannot be directly compared in different subjects. (Pachon.) On the contrary, with the same subject, the study of the variations of evolution of the oscillometric curve, in a given pathological case, furnishes an element of valuable information."

PRACTICAL OSCILLOMETRY.

Criterion of the Method of Oscillation.

Again referring to the original experiment of Marey mentioned before, what then is the reason for the zone of increasing

Brathwaite—Cardiography and Oscillometry 29

oscillations from the maximum to the minimum and to what does it correspond? The answer is that they exactly correspond to the compass of the variable pressure; to the maximal and minimal points between which the arterial pressure ranges.

The zone of increasing oscillations, therefore, under gradually decreasing pressure exactly represents the extent of variable pressure to which the artery is subjected. In other words, the entrance into the increasing zone indicates the maximal pressure and the exit from the increasing zone indicates the minimal pressure.

Practically we proceed as follows: The cuff is adjusted with the entrance of the tube directly over the brachial artery and we pump until the manometer M indicates a pressure which is considerably more than the maximum normal blood pressure, say 200 mms. of mercury. From this point the pump is of no further use. We then cause the pressure to diminish little by little say from 200 to 190, to 180, to 170, to 160, etc., etc., mms. of mercury by acting on valve V. At each of these points we press with the same hand on the stop cock S in order to observe the indications of the oscillometer and record on a suitable graph the maximum and minimum oscillations at that particular pressure. We then repeat the process for a pressure of 10 mms. less than the previous one and so on.

We continue to decrease the pressure and then survey the zone of gradually increasing oscillations during which the observer is enabled to study their characteristics.

In the actual manipulation of the oscillometer it is very important to remember that one should never operate the screw V at the same moment as the separator S. (This will impair the aneroid.) To avoid this error, the operator should manipulate these parts with the same hand, using the other hand to record the amplitude of oscillations at the varying pressures.

CONCLUSIONS:

The electrocardiograph permits of the study of the function of the heart.

The oscillometer permits of the study of the cardio-vascular system.

The oscillometer is more than a sphygmomanometer for while it permits us to determine (1) Systolic pressure, it also allows the study of (2) Diastolic pressure with particular exactitude and (3) the oscillometric tracing gives a graphic record of the exploration of the cardio-vascular system, especially when observations are conducted at several different sites.

Other things being equal from the standpoint of the explored vessel the oscillometric curve translates the value of the cardiac impulse.

All things being equal as regards the cardiac impulse the oscillometric curve translates the measure of the vascular caliber, that is the state of resiliency of the vessels so explored.

On the contrary the study of the respective values of the curves in different individuals cannot be directly compared.

The study of the variations of the curve in the same individual determines the value of the cardiac impulse or, the organic or functional alteration of arterial resiliency as well as the values of the peripheral circulation and the state of the vasomotor nervous system.

A well known authority (Dr. Thomas M. McMillan) has suggested that greater emphasis be placed upon the statistical studies of heart disease from both the electrocardiographic and oscillometric standpoints with the object in view of not only gaining information useful in selection for life insurance but also as a definite contribution to clinical medicine.

Aside from the fact that oscillometry is still experimental, and that, aside from the work of French authors there is not much of moment in the literature, it apparently is a fact that if the French claims can be substantiated by either life insurance or clinical investigation, or both, oscillometry will assume a more important position in diagnostic consideration.

To sum up: In life insurance selection the oscillometer permits us

- a. To understand objectively the value of cardiac impulse and the vascular tone of applicant.

Discussion—Cardiography and Oscillometry 31

- b. To obtain a tracing representative of this value.
- c. To follow it up from year to year.
- d. To follow up the effects of a treatment if same is desirable.
- e. To be the basis for statistical studies which (with a cardiographic record) will permit us to arrive at a prognosis with greater precision than heretofore.

DR. PATTON—The subject of this paper is new to most of us and we have not made much use of this method of testing the efficiency of the circulatory system.

Any confirmatory results that we can obtain with reference to diastolic pressure or the resiliency of the arterial walls should be of value to us. Dr. Herbert Old will tell us something of what he thinks about the use of oscillometry in life insurance.

We had also expected to hear from Dr. Gordon Wilson in connection with this paper, but unfortunately he has been detained by important professional duties in connection with the State Board examinations in Maryland and can not be with us today.

Dr. Old hoped to be able to get in touch with Dr. Smith and possibly to get some of his impressions of the work that Dr. Brathwaite has mentioned. Dr. Old.

DR. OLD—Mr. President and Members of the Association: This paper by Dr. Brathwaite is most germane to the present interest in cardiovascular diseased conditions, especially coronary arterial sclerosis as one of the greatest individual causes of death. This interest is not limited to the insurance companies and the medical profession but is equally predominant among the educated laity. The prominence given in the newspapers to sudden deaths on the golf course, or following a game of golf within 12 to 24 hours, or in supposedly healthy individuals while seated at their desks or attending to their routine business, or while quietly resting or sleeping, causes every thoughtful man in the 50's and 60's to think of a like fate awaiting him.

The enormous increase in the number of heart clinics being established at the instigation of the American Heart Association and the publicity of their results, given out by them in pam-

phlets and bulletins to the medical profession and the laity, are having a widespread effect. In their latest bulletin they state that the incidence of heart disease is 2% of the total population—that is, there are over two million persons in the United States who are handicapped by this disease. From a group study of adult ward and private patients the relative causes were:

Arteriosclerosis	40%
Rheumatism	25%
Syphilis	10%
Various causes	15%
Unidentified	10%

In its last analysis, the normal and harmonious performance of function in the various organs of the body is primarily dependent upon an adequate and proper circulation. Hence the mystery, and in many cases the romance, connected with the heart and blood vessels, as we must study and diagnose internal relations by external relations and signs. This being so, it is up to us to adopt and employ all measures that have proven their worth in the hands of clinicians for the detection of circulatory disturbances or impairments.

The electrocardiograph is a most valuable adjunct in the study of what is taking place in the conduction fibres of the myocardium and the extent of any actual damage that may be present. Dr. Willius, of the Mayo Clinic, states that significant abnormalities are revealed by this instrument in about 70% of the cases of coronary sclerosis. Dr. S. Calvin Smith, of Philadelphia, writes me that he believes in the near future we will be able to detect by this instrument the transitional stage between health and disease in the heart muscle. He further emphasizes the importance of not relying on one tracing in borderline and dubious cases, and in not permitting a normal tracing to contradict your clinical sense and the past personal history. Cases where fatigue, or foci of infection, or over-indulgence in food or drink are thought to be the cause of the abnormalities, should be postponed for further tracings in from three to six months.

Discussion—Cardiography and Oscillometry 33

In other words, we must ever keep in mind that the electrocardiograph is like the sphygmomanometer, the oscillometer, and the orthodiagraphoscope, an additional instrument in our armamentarium, and the evidence obtained by its use has to be considered and interpreted along with the other clinical and instrumental evidences.

Dr. Brathwaite has stated in his paper very clearly the many important uses that can be made of this instrument in evaluating insurance risks, and I thought you might be interested in hearing about our own experience so far with the electrocardiograph and the orthodiagraphoscope at the Home Office.

We began this work during the late summer of 1928 and have examined officers, agents, employees, old policyholders, applicants, and prospects. We have on file about 300 records and Dr. Dewees, is in charge of this work. We realize that this is not sufficient material nor has sufficient time elapsed to be able to make any satisfactory report concerning groups, or prognosis with regard to differentiating the normal changes due to advancing age and those due to beginning disease. However, we have been able to detect undoubted myocardial damage in applicants who had been recommended by our examiners and who were re-checked by us on account of some hypertension, or a past history of rheumatism, or some premature contractions. On the other hand, many cases who, from their insurance history or findings at the time of examination had shown circulatory impairments, were found to be insurable. Many of the arrhythmias were found to be due to some toxic effects from teeth or tonsils, or coffee or tobacco, or fatigue, and these were postponed until the cause had been removed and later tracings had proven satisfactory. In talking with Dr. Calvin Smith, I found that his experience corresponded with ours, namely that under age 40 such toxic impairments showed improvement in about 3 months; after age 40 and up to age 58 in about 6 months; over age 58 they rarely disappeared.

We can also state that all individuals who have undergone this additional study thoroughly appreciated the service rendered and

we have had fine cooperation from our General Agents. They, of course, realize that the case is declined or will not be considered unless this additional study is made, and as the amount of insurance involved is usually large, they present our point of view to the agent and the applicant in most forcible language. In order to give you some visual evidence of the work, I brought over several records with a brief history of the case, and any of you so disposed are welcome to examine them after the meeting.

With regard to the oscillometer, I was very much interested in reading what Dr. Brathwaite had to say, and I am very glad that he is giving so much time and thought to this instrument. The principle of the instrument is very similar to that governing the auscultatory method, but depends upon the oscillations being seen and gauged instead of heard. All of us are aware of the difficulty we have in determining the true diastolic pressure by the mercury manometer. This is largely due to the slight sensibility of the mercury not being sufficient to enable one to compare differences in the oscillations at various levels of pressure. Pachon's instrument has been devised so as to provide for a great as well as constant sensibility at any pressure level. It also dispenses with the personal equation of tone or hearing deafness on the part of the examiner. It has evidently not been used in this country very much, judging by the replies I received from eight prominent clinicians in different sections of this country, but they admitted that they were in no position to pass any judgment on account of their lack of experience. The French clinicians praise it highly and I have no doubt but that in due time it will be given a fair trial in the clinics, hospitals, and at the Home Office of the insurance companies, for from the clinical standpoint regarding hyper—and hypotension and arteriosclerosis, more and more stress is being placed upon the diastolic and pulse pressures, in order to calculate the mean pressure, and I know of no field more fertile for the production of both wheat and tares than that of the evaluation of life insurance risks.

Dr. Brathwaite should receive our heartiest approbation and good wishes in being our pioneer in this work.

Discussion—Cardiography and Oscillometry 35

DR. PATTON—This topic is open for general discussion or comment.

DR. CRAGIN—I want to say just a few words on a little experience which we have had in getting diastolic blood pressures. We have felt for a long time that very few of the Examiners really do what we think they are doing when they take the diastolic blood pressure. We have something like 13,000 Examiners in the field so after a consultation with Dr. Root, we decided that the only way to find out whether the pressure was being taken on the fourth or fifth phase was to include a question in our medical blank asking for both fourth and fifth phases. The answers to those questions were most astounding. I won't give the actual percentage; it is too much of a blot on the fair name of medical science of the United States today, but there are extremely few doctors on our list of Examiners that know what the fourth phase is anyway. Most of them, I believe, leave the question blank, and, therefore, when we come to study diastolic blood pressures on the fourth phase or fifth phase, we want to be pretty sure that our physicians in the field know what they are talking about when they are talking about the fourth or fifth phase. We have found that in the majority of cases the doctor has no more idea of what the fourth phase is than nothing at all.

We have used the electrocardiograph since 1925 and now have about 268 cases which are in the process of analysis. I had hoped to be able to bring them along but was unable to finish the analysis in time for this meeting. However, we hope to have them finished for next year's meeting.

DR. PATTON—Dr. Brathwaite, have you anything to say in conclusion?

DR. BRATHWAITE—No.

DR. PATTON—Functional tests of the circulation have been burning subjects for discussion all through the life of this Association. They occupy a great deal of our thought and time each day of our work.

Feeling the stimulus of what had been done by others, and spurred on by our necessities and readings, a definite study was started some years ago by representatives of the Prudential Medical Department. The next paper will present the results of our work, with some observations upon what has been done.

Needless to say, we realize that much is still ahead of us and that we have to do much even to prove to ourselves the real value of our work. What we have done is offered for the consideration of this Association. We feel that some advance has been made and we hope this Association will agree enough with that conclusion to join in our further efforts, thereby assuring, in the years to come, definite proof of the real value in life insurance selection.

We have been very fortunate in having Dr. Philip V. Wells associated with this work. Dr. Wells' knowledge and training as a physicist and statistician have been of immense value.

The introductory summary will be presented by Dr. Edwin G. Dewis.

DR. DEWIS—Mr. President and Gentlemen: I intend to read a very brief synopsis of our paper, following which Dr. Wells will give a practical demonstration of the flarimeter test.

FUNCTIONAL TESTS OF THE CIRCULATION

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SYNOPSIS.

After referring to the great economic loss sustained by life insurance companies due to increasing mortality from heart disease, to some of the difficulties connected with the use of modern instruments of precision and to the many attempts to devise a satisfactory test for estimating functional efficiency of the heart, we mention three types of tests—respiratory, exercise, and postural, and subdivide the respiratory into two—vital capacity and breath-holding.

Vital capacity is shown to decrease about 1 per cent. per year after age forty and a suggestion is made that it might be of value in selecting overweights. The borderline between normal and abnormal was accepted

as 85 per cent. Through quotations we point out that low vital capacity is correlated with some diseases, that it is of great importance in diagnosis and prognosis in cardiac and pulmonary affections and that when abnormal an explanation is always demanded. In pulmonary and renal disease it runs parallel with ability to hold the breath but such correlation does not exist in normal persons.

A number of authors are quoted giving the normal length of time the breath should be held after full inspiration as forty to fifty seconds and ascribing such ability to myocardial efficiency. One claims it an index of acidosis (1) CO_2 , (2) other acids (decrease in alkali). Another finds a definite correlation between breath-holding time after exercise and mortality.

We conclude from our review of the literature that the voluntary apnoeic pause is a quantitative measure of shortness of breath and can be standardized.

An interesting article by Bürger is discussed in some detail as it shows the danger from intrapulmonary pressures of 40-50 mm.Hg. if maintained for twenty seconds. Strong hearts quickly respond to venous return, while weak ones are not able to restore the systolic pressure to its rest value for over 40 seconds. Intrapulmonary pressures of 50 to 60mm. Hg. caused fainting if continued for 15 to 20 seconds and pulmonary circulation was completely interrupted at pressures of 40mm. Hg. The test was therefore considered too severe for use in life insurance. Neither this nor the cardiorespiratory test were measures of shortness of breath.

Pulse rate had been adopted as the measure of fitness by some, systolic pressure by others, but as no reasons for such selection had been given it is suggested that conclusive comparison between them must be made before any respiratory test with which either was used can be considered properly standardized.

Under exercise it is shown that the heart is the chief factor in the circulation, though its efforts would be unavailing without cooperation of the agencies concerned with peripheral circulation, that the oxygen requirements of the body are in almost direct proportion to the external work performed, that carbon dioxide and hydrogen ion concentration result in increased pulmonary ventilation but that the value of this depends on the ability of the heart to meet the added demand either by increased rate or increased stroke volume. It is also shown that efficiency of the heart is indicated by the volume it can pump in relation to the oxygen requirement of the body and that when oxygen debt exceeds what the individual can carry and make up, excessive breathing and rapidity of pulse result, and further that the deciding factor in severe exercise may often be the efficiency of the coronary circulation.

The need of a standardized exercise test is referred to, and one designed by Master and Oppenheimer discussed.

It is stated that in our experience with functional tests, nervous conditions were the most serious of the disturbing factors, however, the order in which the tests were arranged was that most likely to overcome apprehension and emotion. The hyper-activity of excitement and the hypo-activity of perfunctory performance had to be corrected as far as possible by explanation and persuasion. The great majority could be taught with little difficulty to inhale fully so that expiration time became an accurate measure, with the large orifice of vital capacity and with the small, of ability to hold the breath.

The small orifice changed the whole aspect of the test. The reactions were orderly and sustained making them susceptible to accurate measure-

ment of the reactions due to decreased oxygen supply and accumulating carbon dioxide. Normal subjects show no correlation between blows with the two orifices. Blows with the small orifice were about three times as long as those with the large and might possibly have twice the selectivity, but this remained to be proven by correlating the results with the clinical symptoms and finally with mortality.

The systolic drop (S—) is believed of doubtful value because of the technical skill required, the rapidity with which it occurs and the difficulty of estimating it accurately. It usually was noted within the first fifteen seconds and was not quite as marked with twenty as with 40mm.Hg. pressure.

A portable water spirometer, The Flarimeter, is presented. It is simple in design, accurate, durable excepting the rubber tubing, has a compression air chamber with two orifices, one having 200, the other 36 cc. flow of air per second and an upright removable and protected glass tube marked to register 272mm. of water which is much more sensitive to changes in pressure than its equivalent 20mm.Hg. The pressure is therefore more accurately maintained and observed.

The values of the various findings are discussed and a statement made that the systolic responses require further study before their significance can be known.

The borderline between the normal and abnormal of both vital capacity and breath-holding are shown to be pretty well established and the marked shortening of the blow following exercise leaves little room for doubt that it will be an exceedingly important index of breathlessness due to myocardial inefficiency, pulmonary impairment, or changes in the blood.

It is suggested that when the fact becomes known that judgment of physical condition will be based, to a considerable extent, on the result of effort to perform the test well applicants will be urged by Agents to do their best and the value of the test will be greater. The only possibility of malingering would be in those wishing to be declined, as none would be able to blow beyond their actual ability.

It is shown that the test is terminated by irresistible demands on the respiratory center for restored ventilation.

We advance the opinion that the test would be of definite value in many circulatory, pulmonary, and metabolic conditions, that it gives a much better idea of an applicant's true blood pressure than would one or two readings as ordinarily taken, and that though the time required for its completion might seem long yet this and any additional expense involved would be of secondary importance if it fulfilled its promise of improved selection.

We claim that it is not difficult to perform, can be applied by any doctor of reasonable intelligence after some instruction and practice and is simple for an applicant who has only to learn to take a full inspiration and then blow steadily.

It is mentioned that when the test is always performed in the same way it is not essential to determine all the responses. Analysis of one or more of these would therefore be possible and this would permit combining them with similar responses obtained by other companies who might wish to unite in accumulating data.

Since completing the paper, of which the above is a very brief resumé, we have made an intensive study of Flarimeter reports received from the field and completed in the Home Office. As a result we have decided to simplify the field test by omitting the initial systolic drop.

INTRODUCTION.

The economic loss sustained by life insurance companies through disease of the circulatory system is enormous, and far greater than it should be.

Mortality statistics compiled by the Bureau of the Census for the Registration Area Continental United States show that in twenty-five years, 1900 to 1925, there was an enormous increase in death rate—about 40 per cent.—from organic heart disease. So great has been the increase, which we understand is continuing, that deaths from this have exceeded those from tuberculosis and have advanced to first place in the list of causes.

The facts stated are well known to all in this Association and mentioned only to emphasize the great need of some means whereby diagnosis and prognosis may be improved.

There can be little hope for betterment unless the many efforts being put forth for prevention of circulatory disease such as by the Heart Committee of the New York Tuberculosis & Health Association are notably successful or some method more satisfactory and more available for detecting such impairments is discovered.

The many advances made in recent years because of the introduction of new instruments have rendered diagnosis more certain and increased our knowledge of circulatory defects materially. Much technical skill and experience in interpretation, however, are necessary if reliable results are to be obtained from use of most of the aids to which we have just alluded. They are available to comparatively few because of cost. Insufficient skill and lack of experience in any but large centers would, with rare exceptions, make their use unsatisfactory.

As yet we know of no instrument or test, available to all, which will give definite information about the functional capacity of the cardiovascular system. The discovery of an instrument or test which would reveal impaired function in its early stage would indeed be an outstanding achievement

and of vital importance not only to life insurance but to medicine in general.

The need for such a test is eloquently exemplified by the numerous and varied attempts to devise one. Their number makes complete review of the literature an arduous task and detailed discussion here quite impracticable. They may, however, be grouped under three headings.

RESPIRATORY MUSCULAR POSTURAL

POSTURAL tests are no doubt valuable for estimation of vasomotor tone and ability of the system to rapidly adjust itself. They are not difficult to apply but apparently have not been used in insurance examinations.

RESPIRATORY tests are valuable for quantitating shortness of breath and may be divided into two groups.

1. Vital Capacity.
2. Breath-holding.

MUSCULAR. These include the many forms of exercise that have been used at one time or another. Until recently they have lacked sufficient standardization. The changes in pulse rate and blood pressure are so influenced by personal makeup that very often interpretation of them has to be governed by other impressions, yet some general rules have been formulated for guidance in determining the degree of circulatory efficiency present. Recently a well standardized exercise test has been reported which we think is a real advance.

Unfortunately, repeated application of any test is difficult and often impossible in our work. This is, of course, a handicap, yet the same is true of blood pressure, except that the exertion required and the time consumed in completing a more elaborate functional test adds to the difficulty of using it, which is, however, more than compensated by the added information obtained.

Usually we have to base our judgment on tests which would appear insufficient in number to a clinician. This need not discourage us for has not the same been true of blood pres-

sure? What in all our work has been more difficult to assess, than variations of systolic and diastolic pressures? What took a longer time before its importance in medical selection was fully realized? What more difficult to introduce into life insurance examinations, and yet today it is recognized as a necessary aid in evaluating an applicant. Through accumulation of data we are on our way toward some understanding of its significance, even if we do not yet know the exact cause or causes of the variations so frequently seen. It is but one indicator of the functional fitness of and the strain thrown on heart and arteries. Surely similar variations due to similar causes cannot logically be held against the use of some other tests which may be ultimately found of equal, if not greater value. Nor in our judgment can the taking of longer time to apply a test be considered a real bar, provided it gives valuable information about functional efficiency. This would be especially true if the heart happened to be at fault.

After all longevity is largely a matter of circulatory fitness. Any test which will improve selection through greater knowledge of the degree of this fitness is worthy of most painstaking investigation, particularly if there is reasonable ground for believing that it may be a more reliable index of the functional ability of the myocardium.

We have for some time been investigating the effects of various efficiency tests of the circulation and feel justified in presenting this preliminary report, hoping that as a result of continued study we will later be in a position to supplement it with evidence which will support the suggestions and conclusions to be set forth.

An instrument designed in our office, which has been in use by us for some months, is presented in this paper, with an analysis of the results obtained from its use in the Home Office. We have given similar instruments to certain examiners in the field, and have so far found their experience and records very encouraging. The instrument, which we have called "The Flarimeter" from the Latin word "Flare" (to

blow), will be described in detail. In addition case reports, including Flarimeter tests, with electrocardiograms, obtained in the Clinic of the Beth Israel Hospital, in Newark, will be given.

We wish it understood that as the work is as yet far from complete or conclusive, we will welcome most heartily suggestions from any members of this Association.

2. HISTORICAL.

The vital capacity of the lungs is normally limited, not only by the flexibility, but by the size of the thorax and so the average values for normal subjects have been found to correlate closely with height, weight and surface area. The standard tables* of Myers (1923) by height are more practical than those by surface area, for life insurance purposes, although the latter gives a slightly closer correlation. The normal decrease with age is so small that it may be neglected for adults under forty. After forty vital capacity decreases about one per cent. per year. More extensive series may enable us to determine normal grouping more closely; indeed it is possible that vital capacity might serve as a body build factor which would furnish us a better criterion of overweight than can be determined from height and age alone.

The literature on vital capacity is well covered in a monograph by Myers (1925). He accepts 85 per cent. as the borderline between the normal and the pathological. Abnormally low capacities are most marked in pneumonia, quite marked in cardiac disease, while the classical example has been tuberculosis for the routine diagnosis of which Hutchinson developed the spirometer in 1846. Shepard has used it in the same study successfully for over forty years. For prognostic purposes the vital capacity must be used with caution. A sub-normal value may be due (1) to the permanent result of past disease, such as pleurisy, pneumonia, pulmonary tuberculosis or organic heart disease, (2) to a present acute affection which

*A slightly abbreviated form of this table is given in Appendix 1 of this paper, for reference purposes.

may leave no permanent impairment, or (3) to a chronic condition which must shorten the expectation of life.

In spite of the evidence that low vital capacities are closely correlated with disease, which has been accumulating steadily for nearly a century, there are still some who regard these clinical findings as the exaggerations of enthusiasts. Your 1925 Report of the Committee on Dreyer Measurements stated "The result of this test showed that until a better definition of 'physical fitness' is made, it would not be practicable to use Dreyer's criteria in life insurance examinations." Macleod (1926) stated "At one time it was thought that vital capacity of the lungs was related to their ventilatory capabilities, but for years the determination of this value in patients has been considered unimportant. . . . It has become more and more evident, since Peabody and Wentworth's researches, that a determination of the vital capacity is of great importance in the diagnosis and prognosis of several diseases, including heart disease and tuberculosis." Peabody, who did so much to advance the clinical application of this test, stated in a symposium on respiration (1924) "Vital capacity is the best index of lung ventilation, but the test depends very much on the cooperation of the patient. There is a wide variation in normal standards, and also there is no special diagnostic significance to any particular result. Each result must be interpreted individually, and disturbance in lung capacity must be correlated with the clinical picture presented by the patient. . . . Its greatest value is in heart disease. When there is dyspnea on exertion, it is a sign of cardiac insufficiency. The vital capacity of the lungs in heart disease is an index of pulmonary circulation or interference of pulmonary movements on account of circulatory failure."

Whatever our attitude regarding the possibility of a specific interpretation of vital capacity, there can be little doubt that a subnormal value is presumptive evidence of physiological impairment. If an applicant for insurance cannot expire anywhere near the normal value of air for sex, age and

height, this result in itself demands an explanation, and suggests a doubt as to life expectancy. No extensive mortality studies of this have ever been made, but there is just as much ground for expecting this correlation as there was for that of overweight. The one thing needed to make the measurement of vital capacity practicable clinically is a reliable spirometer which is easily portable, and this we have provided in the "flarimeter".

A notable aspect of the clinical findings in many cases is very great reduction in vital capacity. Yandell Henderson ascribes the reduction in pneumonia primarily to atelectasis. We think, however, that pleurisy, dry or wet, which so frequently accompanies this disease must when present be a material factor in the reduction. In tuberculosis congestion and consolidation in proportion to their extent are again important factors in reduction, but the efforts of nature to restrict motion in the affected area by localized decrease in thoracic expansion cannot be disregarded. Myers calls attention to a very interesting illustration reported by Pratt, of marked recovery in vital capacity in acute myocarditis. It increased steadily during a year in bed from 23 to 74 per cent. of the normal. This was undoubtedly due to improvement in myocardial efficiency, pulmonary congestion being gradually relieved with improved circulation in the lungs and thoracic muscles and brain. Greater and greater effort, therefore, both mental and physical, became possible, so that vital capacity gradually increased. Myers states "In cardiac disease it is believed that the vital capacity is reduced because of a stasis and enlargement of the capillaries in the walls of the lung alveoli which affect the elasticity and expansibility of the alveolar walls." These, including other well known associated factors combine to produce reduction in proportion to the degree of their severity.

In some cases the reduction in vital capacity is accompanied by shortness of breath. Wittich and Polczak (1926) found parallel reduction in vital capacity and breath-holding

time in 139 cases of tuberculosis and in 17 of cardiorenal disease. This suggests a connection of vital capacity with the respiratory center, which will be discussed later, but it is probably more often limited by other factors.

Wiggers (1923, p. 417) points out that the vital capacity is normal in myocardial asthenia, with symptoms of pains in the chest, shortness of breath, etc. Among normals, moreover, there is little relation between the vital capacity and breath-holding time. Jackson and Lees (1929) find this correlation to be only 0.16 ± 0.07 in 100 healthy male university students, and state "breath-holding time at the end of tidal (quiet) expiration must depend chiefly on factors other than vital capacity."

Routine breath-holding tests date from the beginning of the century, although Valsalva's experiment, upon which they are based, is nearly two centuries old. Valsalva studied the changes in the pulse when the intrathoracic pressure is increased. Wiggers (1923, p. 631) gives a beautiful reproduction of a tracing of the effect, taken with optical apparatus, which "indicates that the pulse amplitude becomes smaller at the same time that the entire curve falls, and the dicrotic notch becomes larger. It seems more plausible, therefore, to regard the prolonged rise of intrathoracic pressure as interfering with the venous return and so reducing the output and the arterial pressure." Wesley Mills (1889) describes the influence of respiration on the circulation in considerable detail. He states, "That the respiratory movements do exert in some way a pronounced effect on the circulation the student may demonstrate to himself in the following ways: 1. After a full inspiration, close the glottis and attempt to expire forcibly, keeping the fingers on the radial artery. It may be noticed that the pulse is modified or possibly for a moment disappears. 2. Reverse the experiment by trying to inspire forcibly with closed glottis after a strong expiration, when the pulse will again be found to vary. In the first instance, the heart is comparatively empty and hampered in its action, intrathoracic pressure being so great as to prevent the en-

trance of venous blood by compression of the heart and veins, while that already within the organ and returning to it from the lungs soon passes on into the general system, hence the pulseless condition. The explanation is to be reversed for the second case. The heart's beat is modified, probably reflexly, through the cardio-inhibitory center, for the changes in the pulse rate do not occur when the vagi nerves are cut, at least not nearly to the same extent."

This quotation from an earlier generation shows that the physiologists were well aware of the meaning of Valsalva's experiment, but it remained for a clinician, Sabrazès of Bordeaux, to propose the first breath-holding test in 1902. He instructed the patient to hold the breath as long as possible after quiet expiration, to avoid increasing the intrathoracic pressure. The normal maximum voluntary apneic pause was 20-25 seconds. Mitral insufficiency with complete asystole reduced the pause 5-10 seconds.

Stange of Petrograd, in 1914 recommended testing the ability to hold the breath after a maximum inspiration, to determine the condition of the heart muscle. The normal period he found to be 45-50 seconds. Less than 20 seconds he thought contra-indicated anesthesia. His cases of pulmonary tuberculosis averaged 25 sec., chronic bronchitis 28, emphysema 24, mitral insufficiency 22, mitral stenosis 20, aortic insufficiency 15, chronic myocarditis 17, aortic aneurysm 10.

Meanwhile Yandell Henderson (1914) had been attracted to the time that the breath can be held as an index for acidosis, and as soon as Stange's paper appeared he pointed out that Stange seemed unaware of the parallelism between his results and the degree of acidosis known to be associated with these diseases. Assuming the sensitiveness of the respiratory center constant, the blood stimulus can be ascribed to two factors (1) CO_2 , (2) other acids (or deficiency of alkali). Whenever the alkaline reserve is low, the sensitiveness to CO_2 must be increased, and this is Henderson's explanation of the shortness of breath.

The diagnostic and prognostic value of breath-holding tests was emphasized by McMechan (1922) in an enthusiastic paper assembling an impressive literature, which attracted to the subject Wittich and Polczak (1926). These investigators compared the breath-holding times, by the methods of Stange and Sabrazès, with the vital capacity on 338 normals and on 180 pathological, chiefly tuberculous subjects, showing a very marked parallelism in the percentage reductions. They remark "These tests are not to be looked upon as short-cuts to the diagnosis of diseased states. But careful, intelligent application of breath-holding observations to pathological states of the circulatory, respiratory and renal apparatus is a very promising field, and one which the clinician will find worth while."

It is evident from the literature that breath-holding tests are clinically significant, but the real meaning of the breath-holding time is most forcibly revealed in a test devised by Palcso (1928) in Hungary, modifying an earlier test by Gonczy. The breath-holding time is taken with the patient at rest in bed, then after walking at the rate of about 6km. (3.7 miles) per hour the known length of the ward and back. If the voluntary apneic interval following the walk is reduced below 14 seconds the prognosis is bad. Palcso found a very definite correlation between the results of this test and the mortality of 40 decompensated cardiac patients, which is suggestive. The reduction in the breath-holding time is quite surprising, even to normal subjects. After exercise which seems trivial to them, this time is reduced to less than a third of its normal length. Obviously, voluntary apneic pause is a quantitative measure of shortness of breath, and can be standardized to replace the qualitative descriptions of the past.

The British have been quite alive to the value of the breath-holding test as a measure of breathlessness. Haldane (1922, p. 56), in discussing "soldier's heart," observed that "In these cases 'shortness of breath' on exertion was a common

and prominent symptom. . . . Another prominent symptom was that the patients were unable to hold a deep breath for anything like a normal period, even if they were given oxygen to help." Lewis (1920, p. 13) remarked that "Genuine breathlessness is difficult to simulate. . . . The breath cannot be held for any length of time, and if held the distress is exaggerated. In effort syndrome cases of a moderately severe type who are up and about the breath cannot be held as a rule more than ten seconds." Wilson and Carroll (1919) considered a marked slowing of the pulse or loss of volume when a long breath is taken and held to indicate vagus depressor irritability.

The British Royal Air Force have adopted a breath-holding test of rather severe type for their aviators, and Law (1929) has recently applied it clinically to the Cambridge University crew. The subject after taking a deep breath blows against a closed mercury manometer as long as possible, keeping his intrapulmonary pressure at 40mm.Hg. The pulse is counted in 5 second periods throughout the blow. A blow of less than 40 seconds is considered unsatisfactory, and it should be held at least 50 seconds. The pulse rate should stay constant or rise moderately. A large increase (say to 140) is taken to indicate a venous pooling and is unsatisfactory. Marked physical inefficiency is indicated by a pulse rate which rises to a high level very rapidly (in 10-15 sec.) and then falls to normal or below, during the blow. The French Aviation Service use the test of Binet and Bourgeois, rejecting candidates for high altitude flying who could not hold their breath over 45 seconds after deep inspiration.

The most severe breath-holding test, however, is the "Press-probe" designed by Bürger in Kiel. The subject first takes ten deep breaths in twenty seconds, then blows with maximum force against a manometer for twenty seconds. The systolic pressure is taken six times (1) at rest before (2) at the end of the ten deep breaths in twenty seconds (3) immediately after the intrapulmonary pressure is raised by the blow to 40-50mm.Hg. (4) twenty seconds later (just before ceasing

the blow) (5) immediately after letting go and (6) twenty seconds after resuming free breathing. The prebreathing depresses the systolic pressure from 5-40mm., the compression of the thoracic contents by the increased intrathoracic pressure reduces it still more in asthenic hearts the systolic dropping to values too low to measure. The venous return at first is completely shut off. The pressure in the pulmonary artery must be raised above the intrathoracic pressure by increasing force of the right ventricle before the circulation through the lungs is re-established. This occurs with strong hearts, the systolic pressure rising before the end of the blow to values above the systolic at rest. In every case the systolic jumps immediately on letting go, but asthenic hearts are not able to restore the systolic to its rest value for over forty seconds, while strong hearts produce a powerful surge which often carries the systolic above 200mm.

The heart rate, also responds characteristically to the increased resistance to the circulation through the lungs. In all cases the rate increases in the beginning of the blow. But asthenic hearts, starved of blood, soon shrink in size, a threatening bradycardia sets in which is a challenge to breathe again. If the test is not stopped, collapse is sure to occur in such cases. Among 136 healthy men, chiefly medical students, tested in this manner, 28 collapsed during the test.

Bürger followed the heart during the state of collapse with X-ray observations. About 15-20 seconds after an intrapulmonary pressure of 50-60mm.Hg. was reached, the subject fainted. The collapse occurred earlier, the more rapidly the intrapulmonary pressure rose. Two groups were particularly susceptible. First, those of slender build, steep diaphragms and relatively small hearts; during compression an extreme tachycardia set in. The second group (only three cases) was distinguished by an especial excitability of the vagus system. Mounting venous pressure resulting from the intrathoracic pressure augmented intracranial pressure, which stimulated the vagus, and slowed the heart. In most individuals, on the

contrary, the accelerator predominated. Bürger immediately stopped the test when he observed a rapid fall in heart rate during the blow, as he found that such types were exposed to danger from the increased intrapulmonary pressure. Pressures may reach as much as 140mm.Hg., and this will readily explain many of the accidents which result from sudden extreme effort.

After reviewing the literature, finding that the phenomenon was observed by E. F. Weber (1851) on himself, Bürger presents some very conclusive experiments on dogs which show that the pulmonary circulation is interrupted completely when the lungs are blown up to 40mm.Hg. This agrees with Gerhard's result that the pressure in the pulmonary artery of dogs is from 28 to 36mm.Hg. The continuous carotid pressure curves show that it is only when the right ventricle can force blood through the lungs that a carotid pulse is visible. The curves show in detail all the phenomena observed in man, including the collapse after 18 to 20 seconds when the intrapulmonary pressure is raised to 35-40mm. or over. In these cases the arterial pressure sometimes drops to the zero line. Cutting the vagus produced no essential change in the phenomena.

Electrocardiograms on 50 healthy men were obtained during the compression test by Bürger, showing extrasystoles and occasional intermissions after the blow, with changes in the conduction time, etc. He also describes a number of quite spectacular cases of sudden collapse, on extreme effort, of apparently healthy men. All showed relatively small hearts, and one, a police captain, was put through the test to collapse during the blow. It is obvious that Bürger's test is too extreme for life insurance examinations, but his work elucidates the physiology of breath-holding tests.

There is no need of reviewing the cardio-respiratory test evolved by Dr. Frost, as he has so ably presented it to this Association. It will be considered later in connection with our experiments. Step 2, holding the breath with closed glot-

tis for ten seconds after full inspiration, and step 4, blowing against a closed manometer at 40mm.Hg. intrapulmonary pressure for ten seconds after full inspiration are used to measure the initial drop in systolic pressure which results from shutting off the venous return by increased intrathoracic pressure. The breath is held for about ten seconds in Frost's tests, twenty in Bürger's, so that neither gives a measure of shortness of breath.

Nowhere in the literature have we seen any satisfactory comparison of the clinical value of systolic pressure and heart rate in respiratory tests. Some choose one measure, some the other, but none give any reason for their choice. We are therefore forced to face the necessity of such a conclusive comparison before any respiratory test can be considered as properly standardized. Indeed, it is quite evident from the state of literature on breath-holding tests that only a beginning has been made in an important field of diagnosis. The physiologists have cleared the way recently with remarkable advances in knowledge of the relations, physical, chemical, and nervous, between respiration and circulation, and it now remains to apply this knowledge to the design of respiratory tests of the circulation which will be suitable to the needs of life insurance medicine.

3. EXERCISE TESTS.

When in 1926 one of us, with Doctor Patton, presented a paper to this Association on "The Pulse in Life Insurance," the value of some aspects of exercise as a functional test was discussed. A number of prominent authors of divergent views were quoted and the opinion expressed that "these symptoms and signs (change in pulse rate, and breathlessness) were largely proportional to the general physical condition of the individual. They are the signs of fatigue to greater or lesser extent and we think the degree is largely to be attributed to the condition of the circulatory system." We knew of no well standardized test at that time, and did not include in our paper that the degree of fatigue manifested

would bear a distinct relationship to the amount and rapidity of the exercise. The test—thirty bendings in sixty seconds—was mentioned and reasons given for preferring it. The necessity of gauging it to the individual was pointed out and the fact stated that it must be sufficiently strenuous to throw an actual strain on the heart muscle.

Such were the opinions expressed three years ago. As a result of investigations made in our office since then the present authors see no reason to view exercise as a test with less favor, though a new method of applying it is being tried.

Exercise involves adjustments and metabolic reactions, not mentioned in the paper quoted, and as the changes in pulse rate and blood pressure depend upon these, we will consider them later.

The series of events which occur during exercise are distinctly different from those which take place when instruments are used designed to throw strain on the heart through changes in respiration and intrathoracic pressure. Because of this it seems reasonable to assume that their combined use will give a more complete picture of the ability of the circulatory system and its controls to coordinate, and withstand increased strain. This is our reason for considering both in the tests to be presented.

The differences between them are tabulated as follows:

EXERCISE TEST.

1. Little change in intrathoracic pressure.
2. Increased respiration with gradual return to normal.
3. No interruption in flow of blood into the right auricle.
4. No diminution in pulmonary circulation.

RESPIRATORY TEST

1. Pronounced change continuing throughout the blow.
2. Increase in respiration only after the test and quick return.
3. Marked but temporary interruption of venous return.
4. Marked but temporary diminution.

- | | |
|---|---|
| 5. Lactic acid accumulation with oxygen debt. | 5. Slight, if any. |
| 6. Fatigue. | 6. Fatigue, except of lips, absent. |
| 7. Return of pulse rate to normal delayed in proportion to oxygen debt. | 7. Return of pulse rate to normal more rapid. |

We fear it has been too often forgotten in attempts to estimate cardiac efficiency that upon closure of the aortic valves following systolic discharge the heart is through with arterial circulation until the next systole. From this point were it not for arterial resiliency and vasomotor control seconded by muscular movements and metabolic changes, to mention only two, blood flow would cease.

Pulse rate and blood pressure variations are brought about through many and not one change. Alfred Stengel (1913) stated, "Placed at its center the heart is undoubtedly the chief factor in the circulation, but without the cooperation of other agencies mainly concerned with the regulation of peripheral circulation and with the return of the blood to the heart, cardiac power, however great, must prove unavailing.... The pathological anatomist and clinician alike.... have failed to realize that other factors outside the heart are of almost equal importance in maintaining the circulation.... One should review all of the factors concerned in the circulation outside as well as in the heart itself to determine, if possible, the cause of the overstrain."

In view of the above it seems very probable that, for a time, compensations for cardiac defect may be so thorough that ascertained variations in pulse rate and blood pressure will fail to reveal impairment. This is especially true when moderate exercise—thirty bendings in sixty seconds—is applied to persons under age fifty. On the other hand, an efficient myocardium might so compensate for defects external to it that again, under similar circumstances, the evidence obtained would be of little value. Who can tell how long compensation will conceal an impairment? Every man is a law

unto himself the result of heredity, habits, occupation and personal care. Eventually some link in the chain will weaken and adaptation begin to fail. When that time arrives one or more of the tests being considered may prove very valuable by indicating trouble, possibly well in advance of other evidence.

There is, therefore, a strong urge to develop some method which will detect failing function and no reason for discouragement because it does not at first point unerringly to the heart.

Wiggers (p. 409) says "During muscular exercise the oxygen requirements of the body are increased almost in direct proportion to the external work performed. Not only the consumption of oxygen per minute but also its utilization during the interval of separate pulse beats, i.e., the oxygen pulse, increases (Henderson and Prince). While the attendant changes in blood CO_2 and H-ion concentration results in an increased pulmonary ventilation which makes a larger supply of oxygen available in the lung alveoli, it is obvious that this can be of no avail unless the minute flow through the muscle capillaries is also increased. . . This occurs partly as a result of an increased minute discharge of the ventricles. . . This may obviously be accomplished in either or both of two ways, namely: by an increased heart rate or by an increased systolic discharge. . . During exercise more blood is squeezed from the muscles into the veins and this is the more effective as the supplying arterial pressure is augmented and the amount of blood within the muscle capillaries during relaxation is much increased, owing to the opening up of dormant capillaries (Krogh). To this muscular pumping mechanism must be added the possible diversion of blood from the liver and internal organs, owing to the vasoconstriction of the visceral arterioles as well as the pressor influence of the deepened respiration.

"The increase in systolic discharge thus occasioned by equivalent degree of diastolic distension varies greatly in dif-

ferent individuals, however. This depends to a considerable degree upon the inherent properties of the cardiac muscle to respond. A 'good' heart will cause a considerably increased systolic discharge with slight augmentation of diastolic volume; a 'poor' heart requires a much greater distension in order to give the same increase in systolic discharge. . . . In normal hearts, however, it practically sifts down to the question of previous training or lack of training. On this basis we are able to explain why in trained individuals the increased minute volume during exercise is accomplished more largely by increased systolic discharge, whereas in the untrained normal individuals the systolic discharge increases relatively little and the entire increase in minute volume is accomplished by cardio-acceleration (Krogh and Lindhard)."

It seems reasonable to conclude that when the heart as a result of degenerative change, toxic conditions or nervous influence is no longer capable of responding normally, by increase in systolic discharge, to increased venous return that it will dilate and the burden of increased minute volume be assumed by excessive increase in heart rate.

Henderson, Haggard and Dolley (1927) state that "The efficiency of the heart is nothing else than the volume of blood that it can pump in relation to the oxygen requirement in the body. This applies alike to the athlete, to the man of sedentary habits, and to the cardiac patient. . . . An exertion which would be moderate and would involve no appreciable oxygen debt for the man with a more efficient heart, involves . . . the incurring of such a debt in proportion as the heart is less efficient. An oxygen debt exceeding that which the individual can easily carry and make up, generally induces both excessive breathing and an excessively rapid pulse."

No matter what other factors enter into the adjustments which take place as a result of exercise, it is very evident that in the last analysis the major portion of the burden does fall upon the heart and that marked change in pulse rate and rapidity of return to normal bear some relationship to its efficiency.

Law (1929) says, as a result of his investigation with hard dumbbell exercise for about one minute on members of the Cambridge University crew, that "the pulse rate of a healthy fit man should return to normal in less than two minutes (and that the extent of) the rise should not be more than one of 20 to 30 beats per minute." In our recent investigations we have found this to be true, and in an analysis of 500 accepted male applicants of varying ages, made by one of us, the average rise following thirty bendings in sixty seconds was 28 beats followed by a return to normal within two minutes. We, therefore, attach some significance to rises in pulse rate greater than thirty and to delay in return to the original count beyond two minutes.

The time required for return of pulse rate following exercise depends not only on the efficiency of the myocardium to supply aerated blood to the tissues but also upon the degree of clinical change that has taken place in the muscles and blood. During exercise the volume of blood in the muscles is enormously increased. Haldane (p. 282) says "the immense increase of capillary paths will greatly facilitate the exchange of oxygen and carbonic acid between the blood and the muscle fibers. There must be a great tendency to fall in the oxygen pressure of the blood passing through the muscle capillaries during muscular work. Unless this fall were approximately compensated for by the opening out of new capillaries, it is difficult to see how a sufficient oxygen supply could be maintained, as in all probability the oxygen consumption in a muscle during very hard work is twenty or thirty times as great as during rest." This oxygen supply may be sufficient, with slight exercise, to reconvert the lactic acid into glycogen with formation of CO_2 and water. With exercise more marked and in excess of the person's ability to meet the demand the rate of reversion of the acid is insufficient. Writing on this subject A. V. Hill (1926 p. 93) says "The oxygen intake may attain its maximum and remain constant merely because it cannot go any higher owing to the limitation of the circulatory

and respiratory system. . . . If, however, the exercise be severe the oxygen requirement cannot be met, even when the heart and lungs have attained their maximum activity; if the exercise be persisted in, the body necessarily incurs what we may call an 'oxygen debt.' Were it not for the fact that the body is able to obtain its energy in this way, by using its necessary oxygen afterwards, severe exercise would be impossible in men. . . . In the magnitude of the oxygen debt, that is in the concentration of lactic acid which his muscles can tolerate, we have what we may regard as a man's 'capital.' In his oxygen intake, determined by the capacity of his heart and lungs, we have what we may regard as his 'income.' (Oxygen debt is) small after moderate exercise—even when prolonged—large after violent exercise, even of comparatively short duration. . . . Inside the muscles are alkalies capable of neutralizing the acid, and the hydrogen ion concentration in the active muscles does not rise very far so long as the amount of these alkalies is adequate. . . . The speed of the recovery process depends upon the magnitude of the breakdown from which recovery is necessary. . . . (The) volume of oxygen actually used by the heart is almost equal (per gram) to that required (but not obtained) by voluntary muscle during very violent exercise. The muscle has to stop *within a minute* owing to oxygen want; the heart, however, owing to its better oxygen supply, when the coronary circulation is efficient, is able to keep up an output of this order for longer periods. It would seem possible that the deciding factor in the capacity of a man for severe prolonged exercise may often be the efficiency of his coronary circulation. . . . Quantitatively the phenomena of exhaustion may be widely different, qualitatively they are the same, in your athlete, in your normal man, in your dyspneic patient."

Briefly, exercise calls more blood to the muscles with increased demand for oxygen through formation of lactic acid, the demand being in proportion to both the duration and severity of exertion as is also the quantity of acid produced. To

meet the demand increased effort by the heart and all the controls which enter into the adjustments—respiratory, vasomotor, hormone—is necessary. Upon the adequacy of coordination of these controls will depend, in a large measure, the efficiency of the heart. Should the heart be impaired, no matter how good coordination may be, earlier signs of failure will appear. May we emphasize, therefore, that the symptoms and signs developed are closely related to the existing condition of the mechanisms involved and to the work performed.

We referred earlier in this paper to lack of satisfactory standardization. Master and Oppenheimer (1929) appear to have solved this difficulty by designing an exercise test which, in their own words, "would (1) permit accurate measurement of work; (2) involve only an ordinary every day muscular activity; (3) be simple enough for use in a hospital clinic or in a physician's office." They seem to have accomplished their desire in an admirable way. We have been using it for several months, in our own investigations, with a great deal of satisfaction. Psychological influence, fear, discomfort and embarrassment are reduced to a minimum, which is a great gain as all tend to increase pulse rate. They reported blood pressure readings only, although pulse rate at the radial was also taken.

The patient is required to step up two steps, each nine inches in height, and to step down the same number on the other side, turn around and retrace his steps over the set of steps they have designed. Walking over these is continued for one and a half minutes. The amount of work may be varied by increasing or decreasing the number of steps taken in the given time. They consider that "for satisfactory performance of the test the resting level (of blood pressure and pulse rate) must be reached within two minutes of the termination of the exercise." They chose one and a half minutes as the duration of the test deliberately after much experimentation as a shorter period of more violent exercise was

Functional Tests of the Circulation 59

not so satisfactory. The amount of work per minute in foot pounds is determined by multiplying the number of ascents by the person's weight. Tables for both men and women are given showing the normal number of foot-pounds work which should be performed according to age and weight.

These tables have been adapted for our work by expressing them in terms of number of ascents, which avoids the necessity of calculation by the physician. They provide a tentative standard. Their accuracy is indicated by the deviations of the calculated from the observed results shown in Table 1,

TABLE 1.

Comparison of Observed and Calculated Exercise
Required to Delay Systolic Return Two Minutes.

Master and Oppenheimer's Tables Applied to their own Series of
Normal Subjects.

Normal Subjects.								
	No. of Subjects	Average Percentage	Average Median Difference	Quartiles Deviation Upper Lower (Observed-Calculated)/Calculated	Range High Low			
<i>Males</i>								
			±					
All Ages	59	-0.1	-2.2	8.9	8.4	-7.4	30.5	-19.7
Under 21	20	-0.4	-3.1	8.3	10.2	-7.4	14.7	-14.1
21 and over..	39	0.1	-1.6	9.2	8.4	-8.3	30.5	-19.7
<i>Females</i>								
All Ages	56	0.2	0.7	8.3	7.3	-7.1	21.1	-23.3
Under 21	26	0.2	0.2	8.0	6.2	-9.6	20.4	-23.3
21 and over..	30	0.2	0.4	8.6	5.2	-10.1	21.1	-19.1

on the normals from which the tables were derived. We are indebted to Dr. Master, who so kindly loaned his original records for our comparison. The only drawback in the test is that the steps used are cumbersome. Dr. Master agrees with this opinion, and is cooperating with us. We have taken the matter up with Becton, Dickinson & Co., and have suggested a very convenient design which will soon be available.

Much space has been devoted to muscular exercise. One might almost think that the test, when properly applied, would be sufficient. Undoubtedly it is of great value, yet we believe there is good evidence that supplementing it with another test which throws strain, more directly and quite differently, on the heart, will be of material advantage. The

data resulting from their combined use may point more specifically to the heart. We think so and purpose continuing our investigation along this line, using the Flarimeter for breath-holding and Master's steps for exercise.

4. EXPERIMENTAL.

Two years ago Dr. Frost reported to this Association that the New England Mutual had experienced seventeen deaths in over two thousand risks, selected with aid of their cardio-respiratory test, in the first five years. This was only 75 per cent. (± 16) of the twenty-three (± 5) deaths expected from their general business during the same period. There was one chance in four of a random sample from the general business being at least as favorable, but even so, this brief experience is encouraging. We, therefore, began our investigation by repeating the experiments described by Dr. Frost and Dr. Amiral, confirming their results on ourselves in practically every particular.

Twenty-seven tests on two subjects indicated that the systolic pressure at rest varies systematically from day to day, and by smaller amounts from moment to moment. Changes in the base line exceeding 5 mm. appear significant, when the subject is not disturbed by emotion. The respiratory fluctuations are larger than this in many subjects. The observational error averages less than 2mm. The chief variability to contend with in the interpretation of the systolic responses is that due to the state of the subject. This averages 5mm. in favorable subjects, ranging 20mm. during a test, and in emotional subjects may easily ruin the test. There is no doubt that by sufficient repetition this test yields a definite response to competent examiners, but this does not answer the question of the accuracy of routine results from the field.

An attempt was made to produce sufficient fatigue, by repeating the maximum expiration through the Tycos spirometer, to show a systematic change in the systolic or diastolic base lines, or in the lengths of blow. Two hundred and

eighty-five trials on three subjects showed no evidence of fatigue other than moral, even when the maximum expiration was repeated every thirty seconds for half an hour. The effort is easily adjusted for by the circulatory and respiratory systems, and then the systolic and diastolic pressures during the ten second intervals at rest between the blows, remain steady.

The first Tycos spirometer often stuck at zero if blown at pressures of 10mm.Hg., or less, so we wrote to the manufacturer and they graciously replaced it with their latest model. When this arrived it read 60 per cent. too high in vital capacity, and it now reads about 70 per cent. high. The principle upon which the instrument is based is to shunt a small percentage of the expired air into a piston, the displacement of which is magnified by the pointer on the dial, indicating the volume expired. At the expiration pressure of 20mm., frictional errors are serious. It is not difficult for a normal subject to control the expiration pressure so that the expiration time is an accurate measure of the volume expired. The examiner can measure this time with the second hand of his watch easily to one second, which gives the volume to 5 per cent., closer than the subject can reproduce his vital capacity. A standard orifice and a water manometer, with a small compression chamber to steady the pressure, is all that is needed.

The next point investigated was the size of the orifice, to understand the basis of standardization. A few experiments sufficed to show that nothing was to be gained by using orifices larger than the one selected by Dr. Frost, in which the flow at 20mm. pressure is about 200 cc. per second. But when a smaller orifice is used the whole aspect of the test changes. Not only do the reactions proceed in a more orderly and sustained fashion, so that they become susceptible to accurate measurement, but the results themselves become tests of the physiological response to the oxygen depletion and the CO_2 accumulation in the blood and tissues due to interrupting the inspiration.

Even as small a series as ten has been sufficient to show this fact. These ten normal adult males average 8 per cent. (± 7) underweight by the M. A. tables; in vital capacity, 8 per cent. (± 13) below normal by height, 6 per cent. (± 14) by weight, and 9 per cent. (± 13) by surface area, by the standards of Myers; in blood pressure 3mm. (± 6) below The Prudential 1922 experience, and show no abnormalities of heart or urine. A summary of the results on these ten normal males with five sizes of orifices having rates of flow, at 20mm., ranging from 24 to 185 cc. per second are compared with the vital capacity as measured on the Benedict-Collins spirometer, and with the maximum time of holding the breath, in Table 2.

TABLE 2.

Effect of Size of Orifice on Maximum Length of Blow.
(10 normal males, 22-49 years.)

Spirometer Orifice (cc./sec.)	Av. Maximum Length of Blow (Tm, sec.)	Percentage of Vital Capacity (V) Expired	% Deviations Eliminated by Ratio (Tm/V)
Collins	4-10	100	100
V=3.98 liters			
185	21	98	67
72	46	84	41
48	51	62	40
36	56	51	15
24	62	37	0
0	69	0	-19

It is immediately evident that the full vital capacity is expired through the Tycos spirometer (185 cc/sec.), while through the smaller orifices the flow is too slow to allow the full lung capacity to be expired before the stimulus to breathe becomes irresistible. The last column shows, by statistical argument, that the factor limiting the length of blow is correlated with the factor limiting the vital capacity when the orifice is large, but has little relation when it is small.

In assembling the data on the ten subjects, they were naturally placed in order of age. It was then noticed that the younger men as a rule gave higher vital capacities and shorter blows than the older men. This is clearly indi-

cated in Table 3, again showing that orifices larger than 60 cc./sec. are in the vital capacity class while smaller orifices are in the breath-holding class.

TABLE 3.

Effect of Age of Subject on Length of Blow.		
Subjects 1- 5	Average Age=32 years, Range 22-39 yrs.	
Subjects 6-10	Average Age=44 years, Range 41-49 yrs.	
Spirometer	Av. Age 32	Av. Age 44
Orifice	Average Deviation of Length of Blow	
(cc/sec.)	(with regard to sign)	
	%	%
Collins	10	-10
185	4	-6
72	5	-7
48	-7	7
36	-10	14
24	-13	12
0	-15	16

Vital capacity, depending as it does on mobility of the thoracic walls and excursion of the diaphragm, evidently begins to decrease somewhere between thirty and forty-five years of age, which agrees with the literature, while, apparently, the respiratory center becomes less sensitive to excess acidity.

The reason for the relation between T_m and V , shown in the last column of Table 2, is now clear. Those subjects which hold the breath a long time are just the ones which have low vital capacity and vice versa, so that the ratio T_m/V is high (or low) on two counts at once. Of course, a series of ten is ridiculously small for any such purpose as age grouping, and the regularities are the result of fortunate freedom from disturbing factors, but there can be no doubt that small orifices give tests quite different from large.

The effect of orifice size on the maximum response in systolic pressure is presented in Table 4, on twenty normal adult males. The 36 orifice gives an average maximum systolic response of 27 mm., while the other orifices give smaller responses for almost every subject. Three out of twenty give larger responses on holding the breath, and two give

larger with the 72 orifice, but considering the large personal variability, and the necessarily large errors on observing a sharp peak in systolic pressure which is occurring at the end

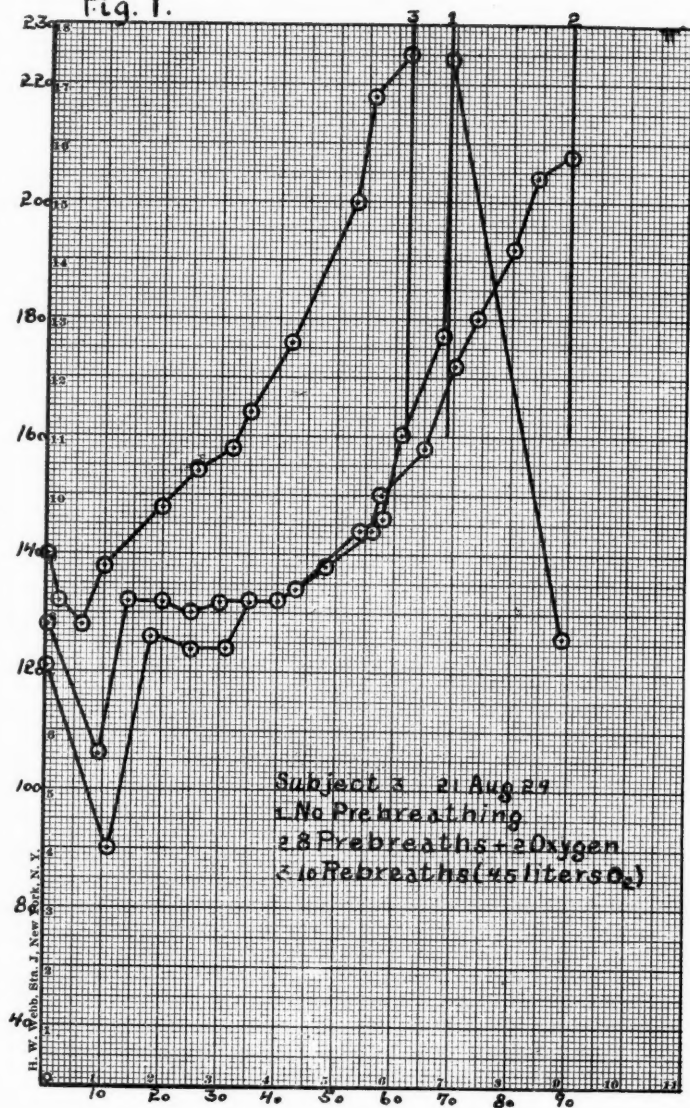
TABLE 4.

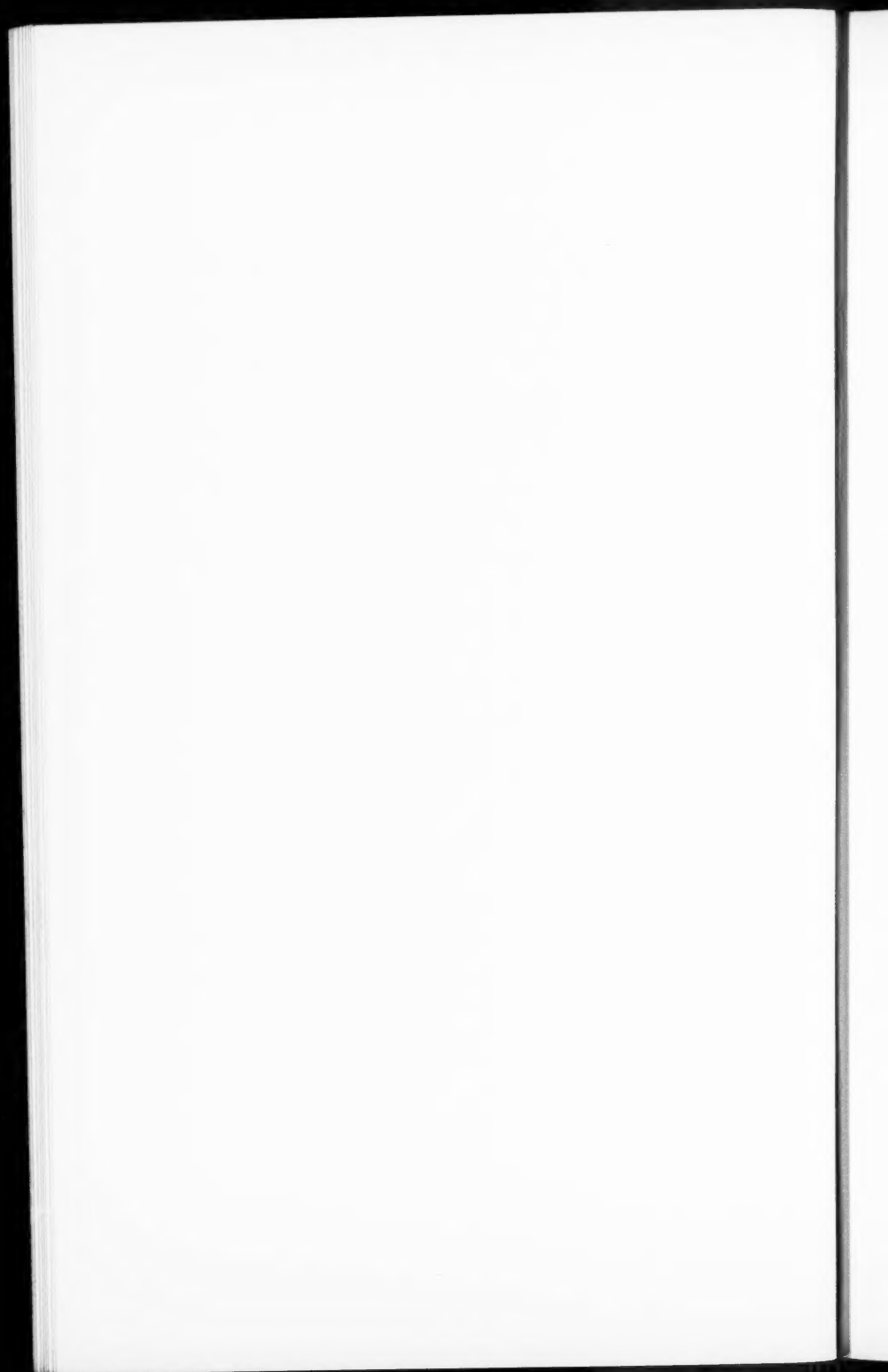
Effect of Orifice Size on Maximum Systolic Response
(20 Normal Males)

Subject No.	Age (Yrs.)	Orifice Rates of Flow (cc/sec. at 20mm.)				
		36	0	24	48	72
		Av. Max. Syst. Response Defect in Response Relative to 36 Orifice (mm.)				
14	22	14	8	-9	-6	-16
25	23	15	-10	0
16	30	25	17	0	-7	5
17	30	30	30	10	4	0
18	32	25	20	0
27	33	31	15	4	9
22	34	5	5	-10
12	37	35	-5	0	5	25
23	38	10	10	0
11	39	18	6	6	0	4
28	39	23	8
10	41	17	15	5	9	13
15	41	25	3	3	-5	7
13	42	51	28
7	42	54	20	16	32	32
8	46	55	20	20	20	35
9	49	32	30	22	4
24	55	30	5	20
26	60	25	6	0
....	23	-5
Average	38.6	27	11	7	6	8
Probable error	—	±2.2	±2	±2	±3	±2.7

of the blow (an uncertain moment, when the muscular tension is more apt to vitiate the reading), in none of these cases are we sure that the results are more than approximately characteristic of the subject under test, or on repetition would be exactly the same. Subtracting the responses with each orifice from that with the 36 orifice, subject by subject, the 36 orifice responses exceed the others by from 6 to 11mm., on the average, which is from two to six times their probable errors. A more extensive comparison might lend

Fig. 1.





greater weight, but the data seem sufficient to justify the choice of the 36 orifice as the tentative standard "small orifice."

Why does this orifice show a maximum response? The only suggestion we can offer is that muscular distension or compression of the thorax is distressing when maintained very long. The distension is soon relieved by the outflow of air through the 36 orifice. We notice that this is just the size at which 51 per cent. of the vital capacity is expired (Table 2). The end of the blow is in the region of tidal breathing, which should be most favorable for resisting the respiratory stimulus, as the element of discomfort is relieved.

To convince ourselves that the responses when blowing through the small orifice are the effect of oxygen depletion and CO_2 accumulation in the blood and tissues, the effects of prebreathing and rebreathing were studied. The best of these tests is shown in Fig. 1, the upper three curves showing the course of the systolic, the lower two of the diastolic pressure. The end of each blow is indicated by a vertical line, numbered to show the sequence. Blow Number 1 is the normal systolic reaction. Before beginning blow 2, eight forced breaths were taken to wash CO_2 out of the blood, then two deep inspirations of oxygen, to prevent oxygen depletion. The curve shows that the systolic response is much the same, but is delayed, as would be expected because of the preliminary acapnia. Before blow 3, ten rebreaths were taken into the spirometer previously filled with 4.5 liters of oxygen, and as expected, the systolic response is much accelerated. Rebreathing has prevented the elimination of CO_2 and so it takes less time for the systolic pressure to increase.

The effects of prebreathing air and rebreathing oxygen on the length of blow through the small orifice are shown in Table 5. The effect of oxygen is more marked on some subjects than others, but the results show that the factor which limits the length of blow through the small orifice is

TABLE 5.

Effect of O ₂ and CO ₂ on Length of Blow through Small Orifice.			
Subject No.	Prebreaths (Air)	Rebreaths (Oxygen)	Av. Increase in Length of Blow
1	10	0	17%
2	7	0	37
	10	0	91
	0	2	102
3	12	0	23
	18	0	27
	8	2	30
	0	2	20
4	10	0	43

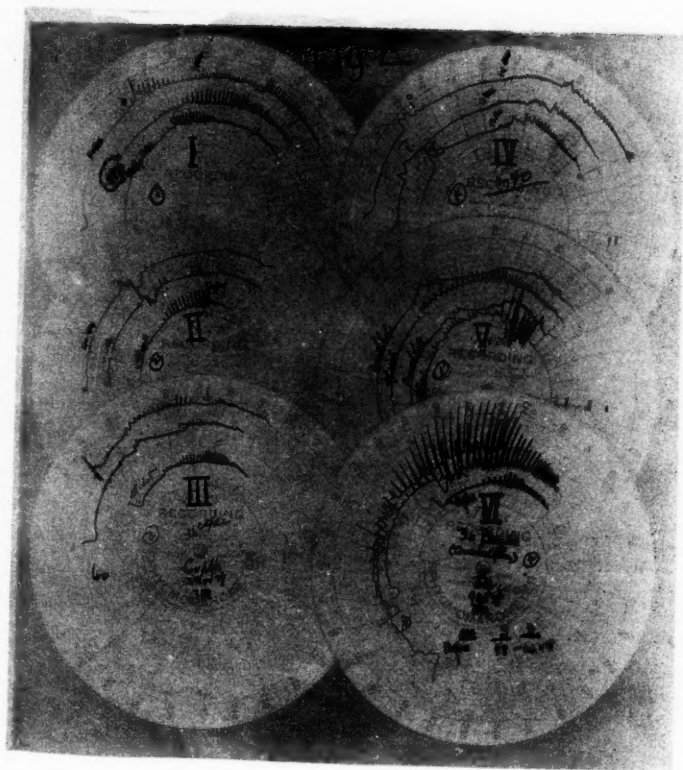
mainly the accumulation of CO₂ in the system, the respiration having been temporarily cut off. The effect of forced prebreathing on the vital capacity as measured by the Tycos spirometer is negligible. Thirty-three trials on five subjects showed an average increase of 8 per cent. when the blow is preceded with ten forced breaths. Seventeen trials on four subjects gave an average increase of less than one per cent. in the vital capacity as measured by the Benedict-Collins spirometer as the result of prebreathing oxygen.

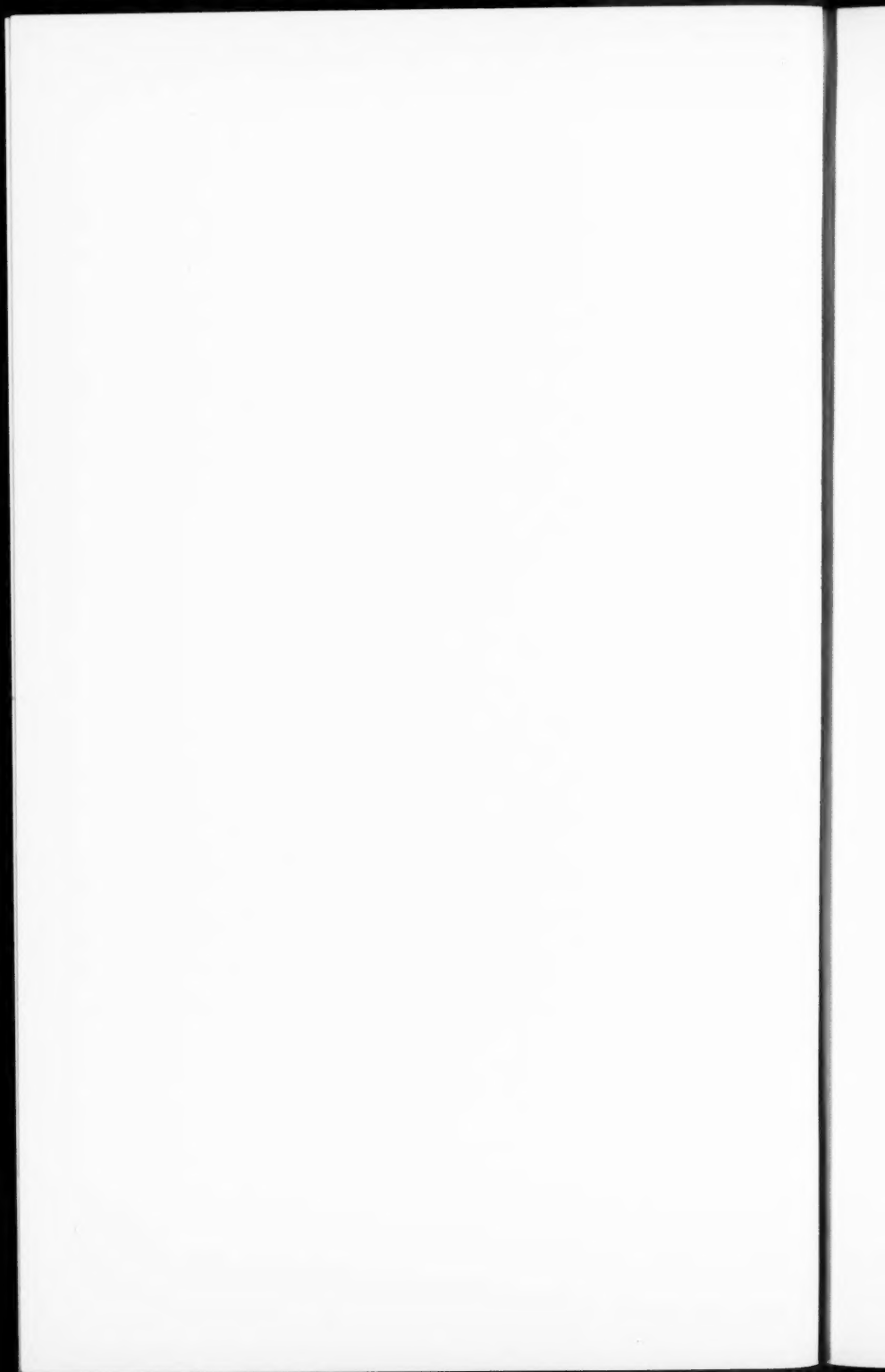
In his published reports Dr. Frost has made little use of steps 2, 3 and 4. Indeed, the prospect of obtaining reliable data from the field is less hopeful in these, because of the technical skill required. In following the systolic pressure when it is dropping abruptly, the sound is often reduced below the threshold of audibility. In step 4 the intrathoracic pressure is increasing, because the intrapulmonary pressure stays constant while the elastic recoil of the lungs is decreasing. The only possible way for the venous return to reestablish itself is for the pressure in the cistern of Keith to rise until it overcomes the intrathoracic pressure. This occurs usually within fifteen seconds, and then the systolic pressure rises from its minimum, quite abruptly at first to its rest value, then steadily above as long as the breath is held. A marked initial drop is also observed in steps 5 to 8 of the Frost test, and in small orifice blows the initial drop is almost as marked as when holding the breath at 40mm.

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The initial drop is too rapid for precise determination, but we have nevertheless attempted a comparison of the values obtained on twenty normal adult males when holding the breath (1) against the glottis, (2) against 20mm., (3) against 40mm., and (4) the initial drop when blowing at 20mm. pressure through the small orifice at 36 cc. per second. The results of six hundred such trials are summarized in Table 6, the column headed 0-G, 0-20, 0-40 and 36-20, referring respectively to the above steps (1), (2), (3) and (4). The probable errors of the median responses are less than a millimeter in pressure, so that there is no doubt that Frost's step 4 usually gives the largest initial drop. This would be expected if the initial drop is due to shutting off the venous return by excessive intrathoracic pressure.

TABLE 6.

Initial Systolic Drop Holding the Breath After Full Inspiration.
(20 Normal Males, 24-56 Years.)

	0-G	0-20	0-40	36-20
Number of Trials.....	179	83	217	123
Median Maximum Drop (mm.)	20	27	34	24
Probable Error (mm.).....	± 0.5	± 0.7	± 0.4	± 0.4
Median Seconds to Minimum				
Systolic	8	8	10	9

When no sound is heard anywhere in the whole pressure range the stethoscope has failed to give true criteria of the pressures in the brachial artery. The Tycos recording sphygmomanometer, however, is well adapted to the study of this point. The record is started well above the systolic pressure and at the proper moment after the first kicks of the pen register the systolic pressure at rest, the signals to inspire and blow are given and a stop watch snapped when the manometer reads the right pressure. The diastolic rises and the systolic falls, reducing the pulse pressure so much that the pen hardly vibrates. This is just what one would expect if the venous return is shut off. A few tracings are shown in Figs. 2-I, 2-V. The sudden breaks toward their ends are where the subject let go, causing a rapid rise in systolic,

and simultaneous fall in diastolic to their rest values. Fig. 2-VI is a tracing after the exercise test, to show the remarkable increase in stroke volume of the heart compared with the amplitude at rest. Such tracings often show graphically the large respiratory fluctuations in systolic pressure which occur in some subjects. Such records have the advantage of freedom from subjective bias, and the first 500 records have shown that the criterion for the systolic is satisfactory. The record often gives larger values for the initial drop than are caught by the stethoscope. In neither case can the true maximum value be caught exactly except by rare accident, so that average values must fall far short of the maximum initial drop. The first trials of some subjects are so ineffective that the systolic pressure does not fall below the base line at all. One can imagine the diagnostic value of such a criterion in the hands of a perfunctory examiner.

5. THE FLARIMETER.

There are two types of respiratory functional tests (1) vital capacity, (2) breath-holding tests. Their only competitors at present are exercise tests which have practical disadvantages. Blood pressure and heart rate cannot be measured when a subject is bending, jumping, running upstairs, pumping up a tire, or working a bicycle ergometer, nor can these exercises be as easily controlled as can maximum expiration through a standard orifice at a standard pressure. All that is needed in addition to a watch, sphygmomanometer, and stethoscope, with which the examiner is already equipped, is an inexpensive, reliable, portable instrument provided with a large orifice for vital capacity and a small orifice for the breath-holding tests. The Flarimeter (Fig. 3) has been designed to meet this need. Its manufacture was placed in the hands of Becton, Dickinson & Company, of Rutherford, New Jersey, at the beginning of the year. The first production model was submitted to us for tests early in February, the second model in March, and eighteen of the

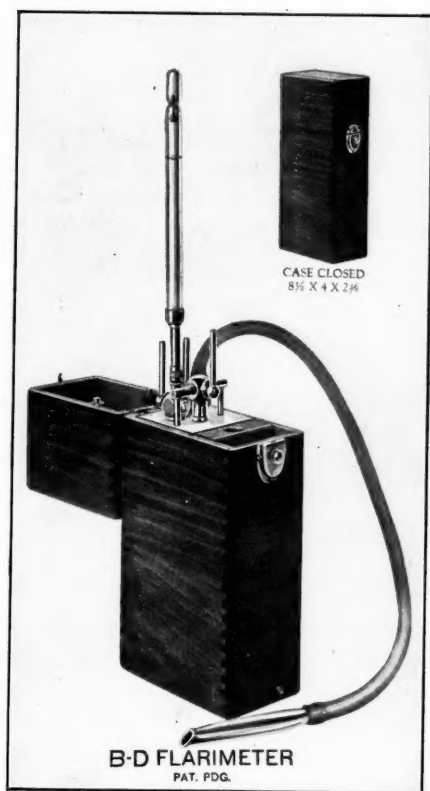
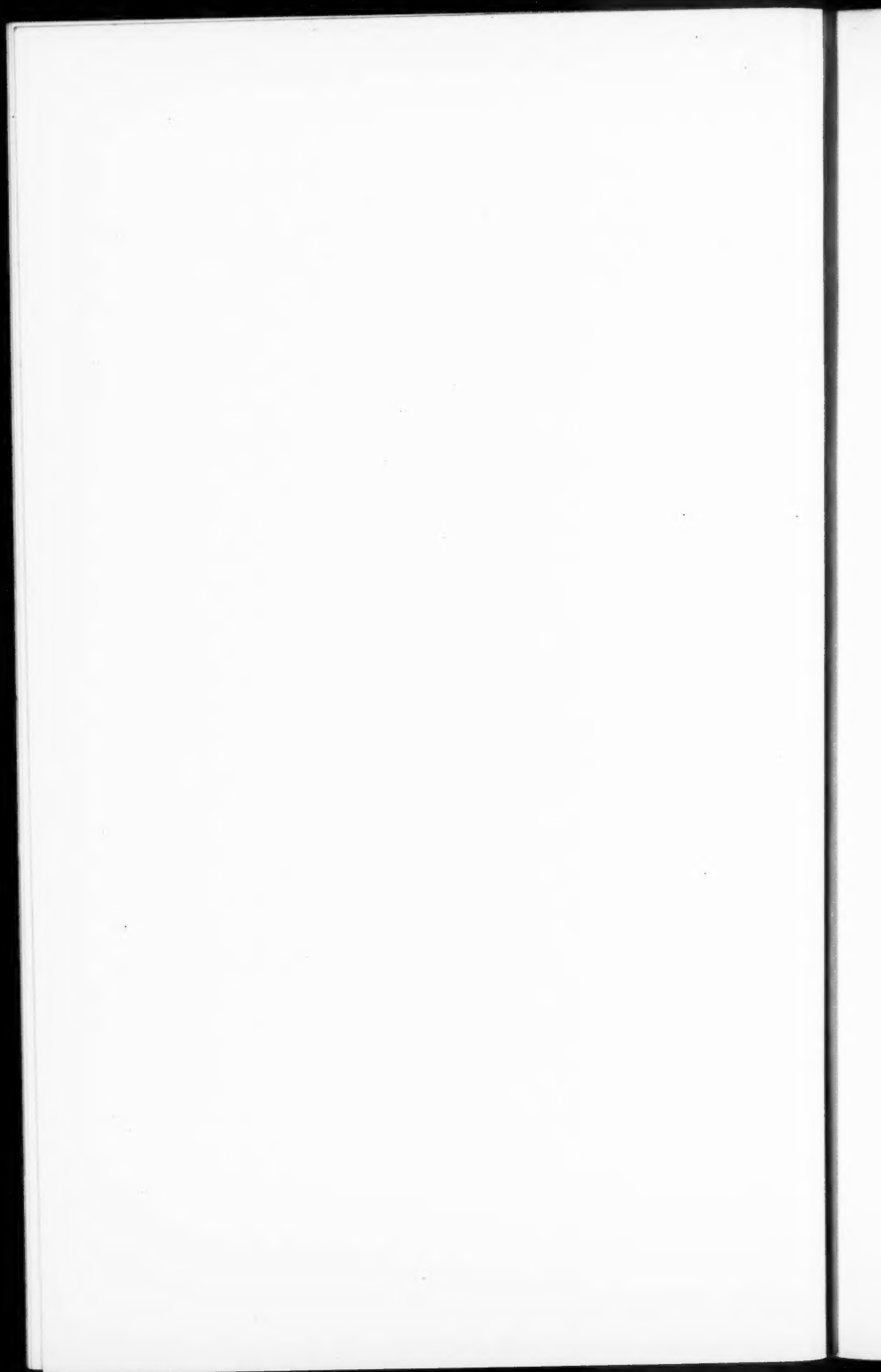


FIG. 3



third production model were tested early in May, before being placed with selected examiners in the field. Our experience with the conditions of manufacture indicates that all requirements will be easily met when the instrument is placed on the market.

The first requisite of a clinical model is portability. This can be obtained without sacrifice of precision with expiration pressures of 20mm. of mercury. This pressure is controlled by a simple water column, which can be reproduced exactly to specification, never requires recalibration, and is sufficiently sensitive without being mechanically unreliable. Moreover, as the air expired from the lungs is saturated with water vapor, which condenses on the interior walls of the instrument, a water manometer is most appropriate. By making the manometer tube removable, the over all height, including the case, is only $8\frac{1}{2}$ inches. Once made correctly, there is no reason why the instrument should not last indefinitely, except for the rubber tubing. The orifices may gradually accumulate dirt, but the constant flow of supersaturated air through them tends to wash them clean. The laboratory model, with a glass orifice, has changed less than 0.3 per cent. in seven months, and only 0.8 per cent. in fourteen months, with no attention whatever. The metal orifices cannot easily be altered by cleaning them with the drill with which they were originally reamed. We have cleaned them many times in succession without changing the flow as much as $\frac{1}{2}$ per cent., the average error of calibration.

The maximum capillary error is less than 2.8 per cent. because the internal diameter of the manometer tube is 4mm. and the water column 272mm. In practice it will not exceed 1 per cent., because it is considered in the calibration. In measuring the vital capacity by the maximum length of blow through the large orifice, the two chief errors are personal to the subject, first, failure to maintain the pressure at the index level throughout the blow, second, failure to fill the lungs completely before beginning to blow. These errors will ex-

ceed all the instrumental errors combined. If the examiner is careful, the length of blow can be estimated easily to within half a second, which is only 2.5 per cent. of a 20 second blow. It must be remembered that the orifice has been calibrated by a method similar to its use in practice. A large reservoir of water, giving constant head, is connected to a bottle having a capacity of 4 liters. The valve is suddenly opened, at the same instant the stop watch is snapped, and the air in the bottle is displaced through the orifice, the rate of flow being regulated by the water manometer. If the subject starts the blow precisely as it was started in calibration, holds the pressure at the index level until the very end of the blow, and the examiner counts the seconds to that instant, the calibration is a straight substitution, and should measure accurately the actual volume expired, except for the differences between the rates of flow of ordinary saturated air at room temperature and of the warm air from the lungs.

Temperature effects may be the most serious of the instrumental errors. If the air from the lungs is at blood temperature (37°C), on cooling to a room temperature of 20°C (68°F) it shrinks 9.4 per cent. in volume, of which 5.5 per cent. is due to gas contraction and 3.9 per cent. to condensation of water vapor. When the room temperature is 25°C (77°F) the shrinkage is 6.9 per cent., when 30°C (86°F) it is 4.2 per cent., and when 35°C (95°F) it is 1.2 per cent. Thus variations in vital capacity as large as 8 per cent. may occur, due to changes in room temperature. Indeed, the seasonal variation in vital capacity which has been observed in the literature may be nothing more than temperature errors. The average shrinkage in practice is probably between 7 and 10 per cent., and if this is corrected for in calibrating the instrument, the temperature errors may be expected to average 2 to 3 per cent. Since the temperature factor is large, and is ignored in the literature on vital capacity, a careful comparison of temperature errors in a Bene-

Functional Tests of the Circulation 71

dict-Collins gasometer type spirometer and in a B-D Flarimeter is shown in Table 7.

The method of calibration has already been indicated, and it is evident that the temperature of the air is that of the calibrating bottle which dominates in heat capacity. Moreover, the air is saturated with water vapor at that temperature, and so by using warm water at 37°C, the conditions in vital capacity measurements are closely imitated. To know the temperature effect it is necessary to vary the room temperature, which is awkward, so that the water temperature has been varied instead. The water temperatures are shown

TABLE 7.

Temperature Effect in Collins Spirometer.
Room Temp. 26°C.

No.	Water	Obs.	-Calculated Shrinkage			Obs.	Obs.	Dev.
Obs. Temp.	Vol.	Dev.	Condens.	Contract	Total	Shrinkage	-Calc.	
°C	cl.		%	%	%	%	%	
26	425							
4	29.2	412 ±1.4	0.7	1.1	1.8	3.1	1.3	.0
2	35.0	397 ±2	2.2	2.9	5.1	6.8	1.7	.4
2	41.7	384 ±.5	4.7	4.9	9.6	10.0	0.4	.9
5	27.4	414 ±1.4	0.3	0.4	0.7	2.6	1.9	.6
5	40.4	381 ±2.8	4.1	4.6	8.7	10.8	2.1	.8
13	47.0	369 ±1.8	6.7	6.5	13.2	13.7	0.5	.8
							Av. 1.3	±.6

Temperature Effect in Flarimeter #17.
Room Temp. 26°C.

		Obs.	Large Orifice.					
		Sec.						
2	29.2	20.6 ±.1	0.7	1.1	1.8	-0.6	-2.4	1.8
3	35.1	19.5 ±.2	2.3	3.0	5.3	4.6	-0.7	.1
3	42.0	18.4 ±.03	4.8	5.0	9.8	10.0	0.2	.4
				Small Orifice.				
3	29.2	111.3 ±.4			1.8	1.9	0.1	.5
3	35.1	107.6 ±1.1			5.3	5.3	0.0	.6
3	42.0	103.7 ±.3			9.8	8.8	-1.0	.4
							Av. -0.6	±.6

in the second column of Table 7, the average apparent volumes observed in the Collins spirometer in the third column, the numbers of observations in the first column. In the third column of the Flarimeter calibrations, the observed lengths

of blow through the orifice, for the bottle capacity of 4090 cc., are given. In the fifth column are the calculated shrinkages due to the condensation of water vapor on cooling to room temperature, and in the next, the contraction of the gas on cooling, according to the gas law. The totals of these, given in the seventh column, compare very well with the observed shrinkages shown in the eighth column, as shown by the ninth column, giving the differences between the observed and calculated shrinkages.

The results show that the Flarimeter has temperature errors about one-third smaller than the gasometer type spirometer. The shrinkages in the latter average 1.3 per cent. more than those calculated from the gas law, which is to be expected, as saturated water vapor shrinks more than a perfect gas. The shrinkages in the Flarimeter, however, are partially compensated for by the increase in viscosity with the temperature, which is about 3 per cent. from 20°C to 37°C. This explains the average superiority of 1.9 per cent. in the Flarimeter calibrations. The thermal expansion of the glass bottle is less than 0.1 per cent., and that of the water is less than 0.4 per cent. in a range of 20°C, and so may be neglected. The random errors average about 0.6 per cent. in both the Flarimeter and the spirometer.

The results of the calibration of the first eighteen Flarimeters ordered for the field are shown in Table 8. Both orifices average about 1 per cent. too small, in flow, the average error being about 1.5 per cent. None of the orifices is more than 3.5 per cent. from the standard flow at room temperature. No correction has been applied for the temperature shrinkage, since it is conventionally ignored in practice, but it should be considered when vital capacity measurements are standardized. Apart from this correction, it is conservative to estimate the instrumental errors at less than 5 per cent., while the clinical tolerance, including errors of personal performance, should be about ten per cent.

Functional Tests of the Circulation

73

TABLE 8.

CALIBRATION OF FIRST 18 B-D FLARIMETERS.

Instrument No.	Large Orifice		Small Orifice	
	Flow cc/sec.	Error %	Flow cc/sec.	Error %
1	199	—0.5	35.9	—0.3
2	195	—2.5	35.3	—1.9
3	197	—1.5	35.3	—1.9
4	195	—2.5	35.0	—2.8
5	197	—1.5	35.3	—1.9
6	201	0.5	36.5	1.4
7	197	—1.5	35.6	—1.1
8	199	—0.5	35.0	—2.8
9	200		35.9	—0.3
10	193	—3.5	35.0	—2.8
11	200		36.2	0.6
12	198	—1.0	36.5	1.4
13	197	—1.5	35.9	—0.3
14	195	—2.5	35.0	—2.8
15	206	3.0	35.3	—1.9
16	197	—1.5	35.6	—1.1
17	199	—0.5	35.3	—1.9
18	199	—0.5	35.9	—0.3
—	—	—	—	—
Av.	198	±1.4	35.6	±1.5
Tolerance		±3.5%		±2.8%

It is interesting to compare the accuracy of these Flarimeters with a Benedict-Collins gasometer type spirometer and a Tycos spirometer used for the Frost test. At 4 liters the Collins instrument reads 4.16 liters, 4 per cent. high, while the Tycos reads off scale, which ends at 6.5 liters. A U. S. Bureau of Standards calibration for the Public Health Service of a Narragansett gasometer type spirometer gave an average error of 1.7 per cent., a range of 6 per cent., and a tolerance of ± 3.2 per cent. The aneroid manometer for controlling the intrapulmonary pressure reads 1.5mm. high at 20mm.Hg., on the Tycos spirometer, an error of 7.5 per cent. We prefer the water manometer to the aneroid, not only because of its greater sensitivity, showing a change in level over twenty-five times the corresponding movement on the aneroid scale, but also because one can always be sure that the reading is approximately correct.

For the purpose of measuring vital capacity alone, the 200 orifice could be even larger to advantage, for there is still a tendency for the values to run slightly under the values obtained with the Collins, which offers practically no resistance to expiration. We have not considered this important but we have used the same orifice as that of the Tycos spirometer, so that steps 5-8 of the Frost test could be performed while taking the vital capacity. For the same reason the pressure drop between the mouthpiece and the gauge has been made about the same (5 to 6mm.Hg.), so that the intrapulmonary pressure is really 25 to 26mm.Hg. when the gauge reads 20. The length and internal diameter of the rubber tubing, therefore, must not be changed, either on the Tycos spirometer or on the Flarimeter, if the calibration of the flow and the intrapulmonary pressure are to be kept standard. The ordinary practice of cutting off a piece of the rubber tubing when it cracks at the connection because of being stretched, or of replacing with nondescript tubing on hand, must be avoided. A new piece of the standard tubing of the standard length must be ordered from the manufacturer. No such care is needed with the small orifice, for the flow is so small that the intrapulmonary pressure is actually that measured on the manometer.

6. COMPARATIVE STUDY.

To study the Flarimeter tests, small homogeneous groups of twenty-two young men and nine young women on the track teams of The Prudential were secured as subjects. All about of an age, and all in excellent condition, they possessed the spirit of cooperation in physical performance so necessary for physiological experiments. A comparison of the cardio-respiratory test with responses to tests performed on laboratory models of the Flarimeter, is summarized in Tables 9 and 10. The first column states the variable measured; the second the median or middle value (one-half the values greater, one-half less); the third the average deviation (with-

out regard to sign) of representative values for single subjects from the median for the group; the fifth and sixth columns the quartiles, which, with the median, divide the frequency distribution in quarters (so that one-half the values fall within, one-half without the quartiles); and the last two columns show the range of variation for the group. Repeated trials of the measurement fall within ranges, the medians of which are shown in the fourth column.

The entire group of males give values for the initial drop in step 2 which are greater than 10mm., and so are normal by the criterion of Dr. Frost. Six of the twenty-two, or 27 per cent. give initial rises in step 3 higher than 20mm., which he considers normal. One subject gives no initial drop to step 4, which should be greater than 10mm. to meet his normal criterion. In at least one out of the four steps 5 to

TABLE 9.
COMPARATIVE TESTS ON 22 PRUDENTIAL MALE TRACK ATHLETES.

Variable Measured	Median	Average Deviation	Repeated Trials %	Median Range of Quartiles			
				Upper	Lower	High	Low
Age (years) _____	21	± 1.5		22	21	23	18
% of Normal Weight _____	100	± 6.5		103	95	106	80
% of Normal Vital Capacity (Collins Spirometer)							
by Height _____	100	± 6		106	94	119	81
by Weight _____	103	± 7		109	96	119	90
by Surface Area _____	98	± 6		104	94	111	85
Excess Blood Pressure (mm.Hg.)							
Systolic _____	1	± 9		11	-6	22	-20
Diastolic _____	-10	± 8		-2	-16	9	-31
Cardio-Respiratory Test—Systolic Responses (mm.Hg.)							
Step 2. Maximum Drop _____	28	± 10	21	42	18	51	14
Step 3. Maximum Rise _____	16	± 8	44	20	8	40	Neg.
Step 4. Maximum Drop _____	34	± 8	32	44	30	N.S.	Neg.
Step 5. Maximum Rise _____	33	± 11	55	44	25	61	2
Step 6. Maximum Rise _____	38	± 11		40	25	64	2
Step 7. Maximum Rise _____	35	± 10		48	27	54	14
Step 8. Maximum Rise _____	33	± 7		42	28	60	16
Trend of Base-Line _____	-1.2	± 7		5	-6	11	-17
Range of Base-Line _____	12	± 5		18	10	34	6
Flarimeter Tests—Systolic Response (mm.Hg.)							
Max. Initial Drop (S-) _____	28	± 12	21	38	16	42	10
Max. Final Rise (S+) _____	40	± 11	20	60	30	70	20
Seconds to Rise 20mm. (T20) _____	45	± 8	11	48	35	76	19
Seconds to Rise 30mm. (T30) _____	50	± 7		54	44	75	32
Seconds to End of Blow (Tm) _____	52	± 10	13	64	45	81	32
Flarimeter Tests—Diastolic Response (mm.Hg.)							
Max. Final Rise (D+) _____	40	± 10	25	48	28	74	22
Max. Drop After (D-) _____	18	± 6	72	25	12	38	8
Seconds to Rise 20mm. _____	18	± 9	17	26	14	54	6
Seconds to Rise 30mm. _____	42	± 8	29	48	33	68	30
Seconds to End of Blow (Tm) - Difference (S+ - D+) _____	50	± 13	6	64	38	81	26
Excess Rise _____	0	± 9		8	-9	32	-21

TABLE 10.

COMPARATIVE TESTS ON 9 PRUDENTIAL FEMALE TRACK ATHLETES.

Variable Measured	Median	Average Deviation	Median Range of Repeated Trials		
			%	High	Low
Age (years)	19			22	17
% of Normal Weight	95	± 6		112	86
% of Normal Vital Capacity (Collins)					
by Height	97	± 10		119	83
by Weight	104	± 9		119	92
by Surface Area	106	± 9		121	93
Excess Blood Pressure (mm.Hg.)					
Systolic	-2	± 5		9	-9
Diastolic	0	± 3		7	-7
Cardio-Respiratory Test—Systolic Responses (mm.Hg.)					
Step 2. Max. Drop	18	± 9	67	44	8
Step 3. Max. Rise	16	± 7	25	28	4
Step 4. Max. Drop	30	± 10	37	N.S.	20
Step 5. Max. Rise	26	± 7		34	5
Step 6. Max. Rise	24	± 8	64	36	3
Step 7. Max. Rise	22	± 6		30	2
Step 8. Max. Rise	25	± 5		34	10
Trend of Base-Line	2	± 3		10	-4
Range of Base-Line	12	± 4		18	4
Flarimeter Tests—Systolic Response (mm.Hg.)					
Max. Initial Drop (S-)	25	± 5	20	28	7
Max. Final Rise (S+)	86	± 6	17	44	20
Seconds to Rise 20mm. (T20)	30	± 9	27	52	18
Seconds to Rise 30mm. (T30)	38	± 10	21	56	22
Seconds to End of Blow (Tm)	41	± 6	20	56	33
Flarimeter Tests—Diastolic Response (mm.Hg.)					
Max. Final Rise (D+)	36	± 10		66	20
Max. Drop After (D-)	11	± 6		26	6
Seconds to Rise 20mm.	20	± 8		38	8
Seconds to Rise 30mm.	27	± 6		44	16
Seconds to End of Blow (Tm)	35	± 5		57	30
Excess Rise (S+—D+)	0	± 11		16	-22

8, which are repeats, there is a normal response (final rise between 20 and 50mm.) in every one of the 22 males, although ten (45 per cent.) fail in at least one of these steps. Fourteen (16 per cent.) of the eighty-eight steps are outside the normal range, so that there is one chance in six of a faulty performance of such steps in normal subjects. Too much weight must not be attached to minor differences in the tests. According to Dr. Frost, the systolic base-line to be normal must not fall more than 5 nor rise more than 10mm., yet nine (41 per cent.) of these subjects do not meet this requirement, and eight of them give an "abnormally falling base-line." The reason is obviously nervousness at the start.

Of the tests on nine female track athletes on the Home Office staff, one in step 2, three in step 3, and fifteen in steps 5-8 are outside the normal ranges. All nine pass step 4 sat-

isfactorily, and the trend of the base-line is within normal limits on all. The Median response to steps 5-8 is 24mm. for the girls against 34 for the boys, so that it is reasonable to assume that the final rise is not as high for the girls. A lower limit of 10mm. for girls in place of the 20mm., fixed by Dr. Frost, would reduce the abnormally low responses from fifteen to four.

In the Flarimeter tests (small orifice) the smallest range between repeat tests is in "T20" (the expiration time required for the systolic pressure to rise 20mm. above its rest value) which is 11 per cent. for the boys, 27 for the girls. The lengths of blow are also fairly well reproduced, the median ranges between repeats being 13 per cent. and 20 per cent. for boys and girls, respectively. Even the maximum rises in systolic are much better reproduced than the corresponding responses in steps 5-8 of the Frost test (median ranges 20 per cent. and 17 per cent. vs. 55 per cent. and 64 per cent., respectively). The reason for the superior reproducibility of the small orifice test is probably its longer duration. The subject settles down to a steady, easy blow, the nervous factor is reduced to a minimum, and the more gradual rise in systolic permits greater accuracy.

The reproducibility data on the diastolic reaction are too meager to have much weight, but they do not indicate that the diastolic pressure would give better results than the systolic. The most interesting result of the diastolic curves is shown in the last row of the tables, the differences in the maximum rise of systolic and diastolic. The average and median differences are negligible for both the girls and boys, showing that the equality of the median rises is not accidental. In other words, the pulse pressure at the peak of the blow is just the same as at the beginning, although both systolic and diastolic pressures have risen, 40mm. in the boys, 36 in the girls.

It thus appears that the small orifice test has succeeded in steadily increasing the mean arterial pressure without mus-

cular fatigue. By increasing the intrathoracic pressure, and so reducing the venous return, and by suspending the respiration, and so producing an increasing concentration of CO_2 and decreasing oxygen in the blood and tissues, which in turn stimulates vasoconstriction, the heart is forced to pump faster at a higher pressure. The diastolic pressure rises 20mm. above its rest-value (diastolic T20) sooner than the systolic does because during the first ten or fifteen seconds the systolic has dropped a considerable distance below its base-line while the diastolic is rising. The final rise in systolic pressure, therefore, starts from a level not much above the diastolic, while the latter begins its final rise from a point usually above its base-line. The contrast between the initial and final responses is shown by the pulse pressure, which is reduced almost to zero during the initial drop, but has resumed its rest-value during the final rise. It is evident that the initial reactions represent a momentary state of circulatory failure, while the final responses characterize steady states, temporary adjustments to unusual demands. Therefore, there should be more time for momentary nervous disturbances to subside, and so the final responses should be less dependent upon the nervous factor.

A comparison of the final rise in systolic pressure with the Flarimeter (small orifice) with that in steps 5-8 of the Frost test (large orifice) shows that the percentage ratios of the maximum responses in the two types of test on the 22 male athletes deviate from their median by 23 per cent. on the average, whereas an average deviation of 35 per cent. would be expected if the responses in the two tests were independent. Proportional deviations averaging 26 per cent., therefore, have been eliminated by taking ratios. The percentage ratios of the maximum response in the large orifice tests (the largest rise in steps 5-8 of the Frost test) to the median, for each subject, have an average deviation of 13 per cent., which can be ascribed only to failure to reproduce the response on repeating the test. There remains of the 23 per

cent. above only 19 per cent. to be ascribed to systematic differences in the nature of the small and large orifice responses which must give rise to independent deviations in different individuals. More than half the variations (in this small homogeneous group) of the final systolic rise with the small orifice are proportional to those with the large orifice, in contrast to those in length of blow, which run more opposed than parallel.

The vital capacities as measured by the Flarimeter average 0.18 liters, or 4 per cent. less than on the Collins spirometer, while the average difference without regard to sign is ± 6 per cent. This agrees with ± 7 per cent., the result on the first ten normal subjects. Taking differences (spirometer-Flarimeter), the average deviation is no larger than that of the spirometer alone, showing at a glance that about half the deviations are parallel in the two instruments, presumably characteristic of the subjects. The average difference between the maximum and next best of four trials is 2 per cent. with the Collins, 3 per cent. with the large orifice, showing the reproducibility of the vital capacity measurements is not quite as good with the Flarimeter as with the best spirometers. The difference is only 1 per cent., less than the superiority of the Flarimeter in temperature errors, and small compared with the tolerance of 10 per cent. which must be considered necessary in routine vital capacity measurements.

The athletes expire on the average 43 per cent. of their vital capacity through the small orifice, which is below that for the first ten subjects (51 per cent.), as would be expected from their youthfulness (av. 21 years vs. 38 years). The average deviation is ± 23 per cent. of the average ratio (43 per cent.), even greater than 21 per cent., the value expected if the lengths were independent, and this also confirms the indications from the first ten subjects that length of blow with the small orifice is uncorrelated with vital ca-

capacity. Jackson and Lees (1929) have found no appreciable correlation between breath-holding time and vital capacity.

The average difference between the maximum length of blow and the next best (by the same subject) is 9 per cent. with the small orifice, three times that with the large orifice. Now this failure to reproduce the length of blow cannot be due to observational error, which is much less, proportionately, than with the large orifice, but must be due to actual changes in the subject from moment to moment, and such fluctuations would be expected to accompany selectivity. Since the random deviations of an individual from his average are uncorrelated with the variations among the subjects, the possible selectivity is measured by the square root of the difference of the squares of the average deviations, and this is 5.7 per cent. for the Collins, 8.5 per cent. for the large orifice, and 17 per cent. for the small orifice. Thus, correcting for reproducibility, there is still twice the possible selectivity with the small orifice that there is with the large orifice. Whether or not these larger variations are truly selective remains to be proven by correlating them with other clinical symptoms and finally with mortality experience.

Flarimeter tests on eighty-eight normal adult males are summarized in Table 11, and the frequency distributions of the responses are shown in Fig. 4. The values for forty-one of the subjects (including the twenty-two athletes), under thirty years of age, are given below those for all ages, to indicate the age effects. The groups are normal in weight and vital capacity. In blood pressure they are slightly under the Prudential 1922 experience, probably because less time was given in the field for the nervous factor to subside. The smallest variability in the Flarimeter responses is in vital capacity, which gives an average deviation of single subjects from the median of about 9 per cent. The corresponding variabilities of T20 and Tm (the length of blow) are 20 per cent., of the final rise (S+) 28 per cent., and of the initial drop (S-) about 50 per cent., of the respective

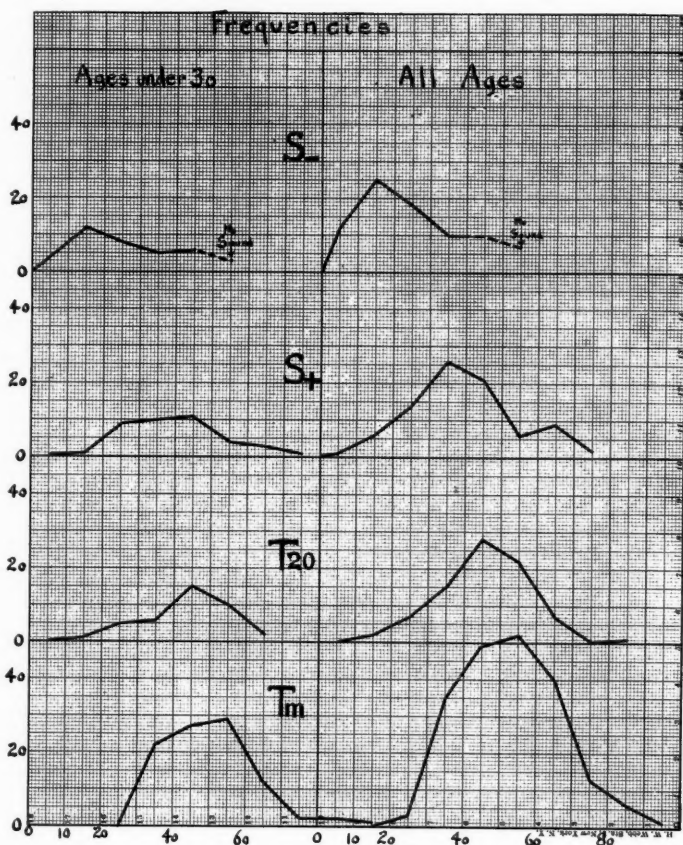
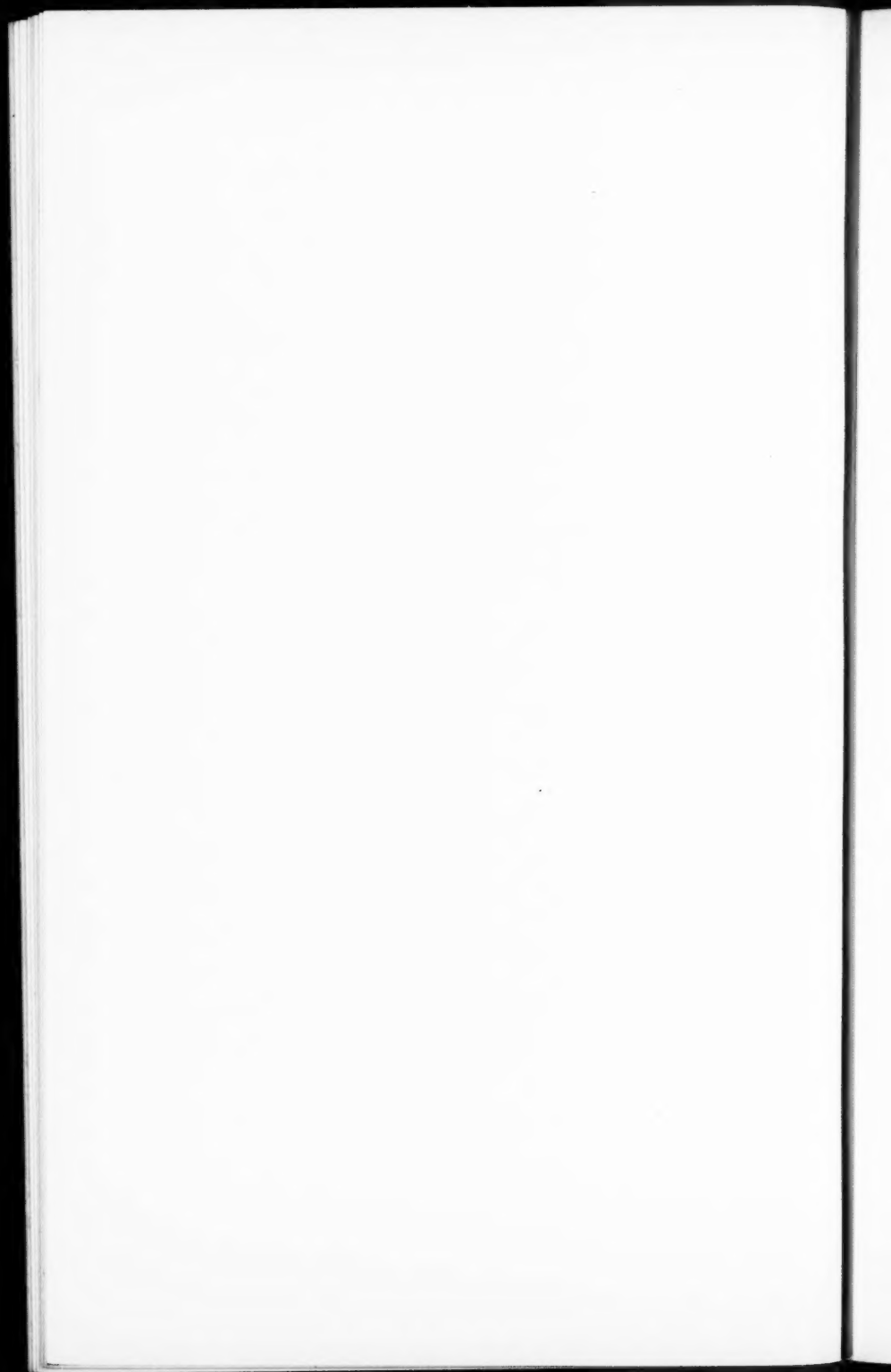


Fig. 4



median responses. These results entirely confirm those on the athletes; probably T20 will be the most selective, when allowance is made for individual reproducibility.

The upper and lower quartiles indicate the limits within which one-half the cases fall, so that the odds are against a normal response falling outside these limits. The entire group covers quite a range of values, but this is usual in such nondescript normals. Heart and urine examinations revealed no significant impairments. The letters "N. S." indicate that the systolic had dropped below the auditory threshold.

Tentative normal standards are set up in Table 12 for the five Flarimeter responses. The odds are taken to be against vital capacities (V) below 90 per cent. being normal, against a systolic drop (S—) less than 15mm.Hg. being normal, and against a small orifice blow shorter than 45 seconds being normal. The odds are against a systolic rise (S+) being normal which is less than 30mm.Hg., or greater than 50. These limits (S+), however, must be considered in relation to the length of blow (Tm). The subject must be given credit for the will power shown. If the length of blow (in seconds) is not at least 10 greater than the systolic rise (in mm.)

TABLE 11.
FLARIMETER TESTS ON 88 NORMAL ADULT MALES.

Variable	Ages	No. of Tests	Average De-		Quartiles		Range	
			Median	viation	Upper	Lower	High	Low
Age (years)	18-62	88	30	11	43	22	62	18
	18-28	41	22	2.2	24	21	28	18
% of Normal Weight	18-62	88	99	7.7	104	90	123	73
	18-28	41	98	6.3	103	89	113	80
% of Normal Vital Capacity	18-62	246	100	8.8	106	90	122	76
	18-28	120	101	7.3	108	94	119	80
Excess Blood Pressure								
Systolic (mm.Hg.)	18-62	528	—4	9.3	6	—10	24	—30
	18-28	246	1	9.2	8	—8	22	—22
Diastolic (mm.Hg.)	18-62	264	—5	7.5	0	—12	17	—29
	18-28	123	—9	6.6	—1	—15	10	—29
Systolic Drop (S—)	18-62	82	26	13.2	43	16	N.S.	4
	18-28	38	22	13.7	42	16	N.S.	6
Systolic Rise (S+)	18-62	85	40	11.4	49	30	74	10
	18-28	39	40	11.3	48	30	74	20
Sec. to Rise 20mm. (T20)	18-62	82	45	9.1	54	37	83	20
	18-28	39	44	8.5	53	36	63	23
Length of Blow (Tm)	18-62	199	54	10.8	62	43	91	21
	18-28	95	47	10.3	58	39	91	31

Note: "N. S." signifies "no sound," the systolic falling below the audibility threshold.

TABLE 12.
ABNORMAL TESTS ON 88 NORMAL MALES.

			V (%)	S- (mm.Hg.)	S+ (mm.Hg.)	T20 (sec.)	Tm (Tm-S+) (sec.) (sec.-mm.)
Tentative Normal Standards							
Quartile Limits							
Lower			90	15	30	35	45 10
Upper					50		
Pathological Borderline							
Lower			85	5	20	25	40 0
Upper					70		
Normal Cases Excluded by Tentative Standards							
Ages 18-62			2%	1%	5%	6%	5%
18-28			2%	None	2%	7%	7% 3%
Abnormality	No.	Age	Responses				
V	54	19	80	16	30	43	55 13
Tm	105	19	100	6	26	39	37 13
(Tm-S+)	59	21	112	12	58	45	52 -4
T20, Tm	64	21	107	8	40	23	32 4
Tm	104	21	108	N.S.	45	33	36 4
(Tm-S+)	74	22	119	20	56	36	47 -4
S+	43	24	118	22	74	44	58 16
T20	38	27	86	14	44	24	58 14
T20	31	28	—	14	46	23	53 7
(Tm-S+)	100	34	115	36	68	47	55 -13
T20, Tm	19	36	94	14	26	20	38 12
S+	23	38	93	9	18	—	66 46
V	11	39	81	8	38	44	45 7
S-	10	41	—	4	22	40	45 23
S+	7	42	—	24	72	50	70 29
S+	13	43	83	—	10	—	43 33
T20	34	49	97	20	70	22	54 3

in the most favorable blow, the odds are that the subject is hyperexcitable. The pathological borderlines are to be similarly interpreted. For example, since only 2 per cent. of the normals have vital capacities below 85 per cent. (when corrected for age by adding to the observed percentage 1 per cent. for each year over forty), the chances are only one in fifty that a vital capacity below 85 per cent. is normal, etc.

The actual responses of all the subjects which show any abnormality in the Flarimeter tests are given in Table 12. Five millimeters may be too lenient a lower limit for the systolic drop. Dr. Frost chose 10 for his step 4, but in view of the technical difficulties with this response we have preferred to discount their pathological significance in this manner. If 10 were chosen, five more subjects would have to be classed as pathological. Ten of the older subjects would have pathological vital capacities if they were not given the benefit of the normal decrease with age. Note that both the subjects giving a systolic rise above 70 show no irritability when they are allowed 1mm. for each second of the blow

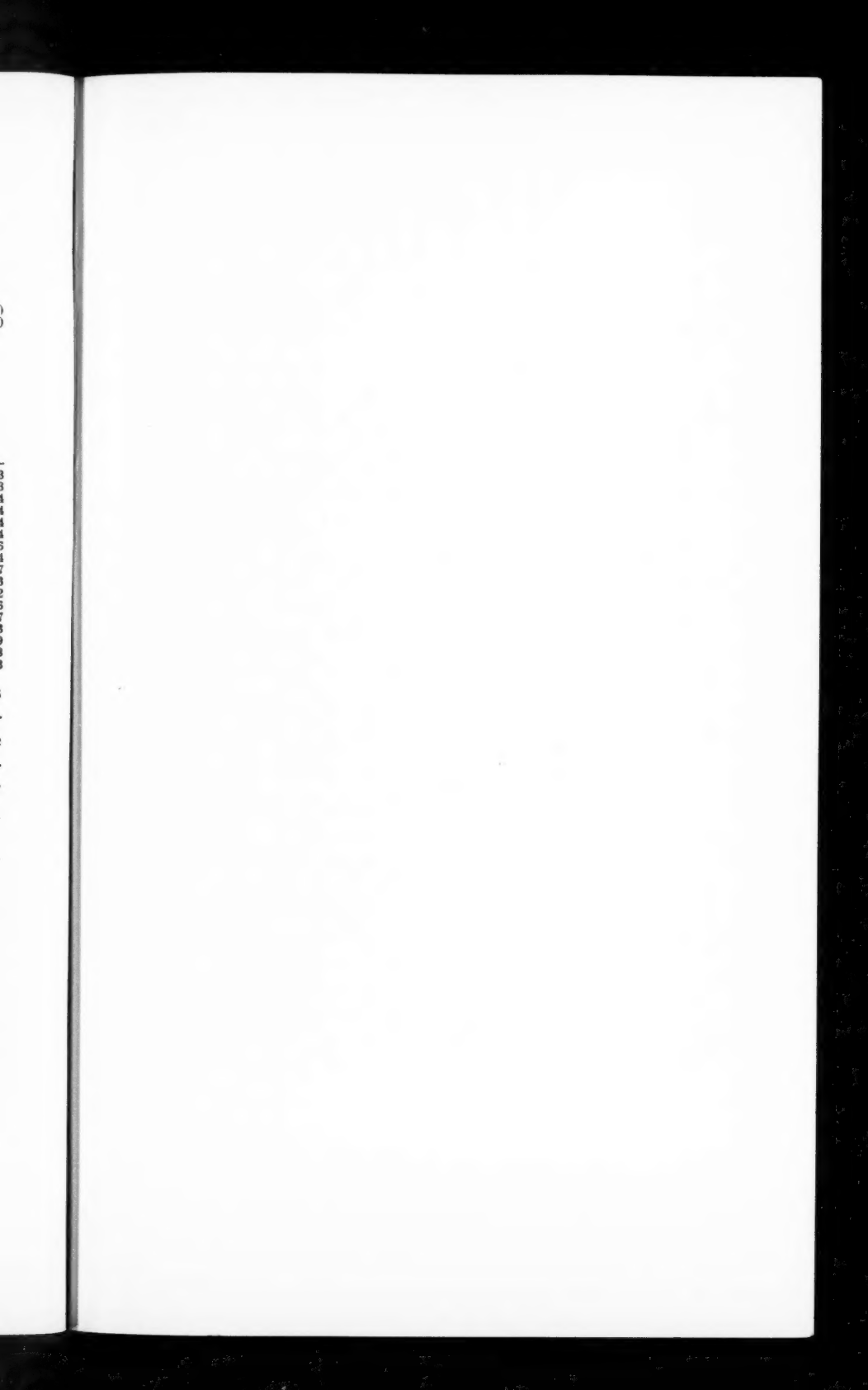


Fig. 5.
Systolic Pressures in Flarimeter Tests

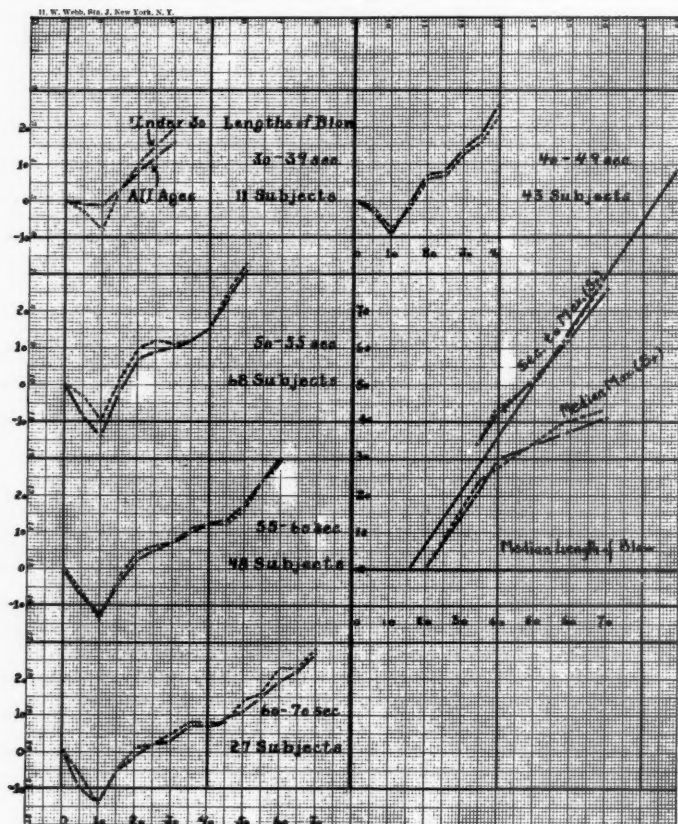




Fig. 6.

Diastolic Pressures in Flarimeter Tests.

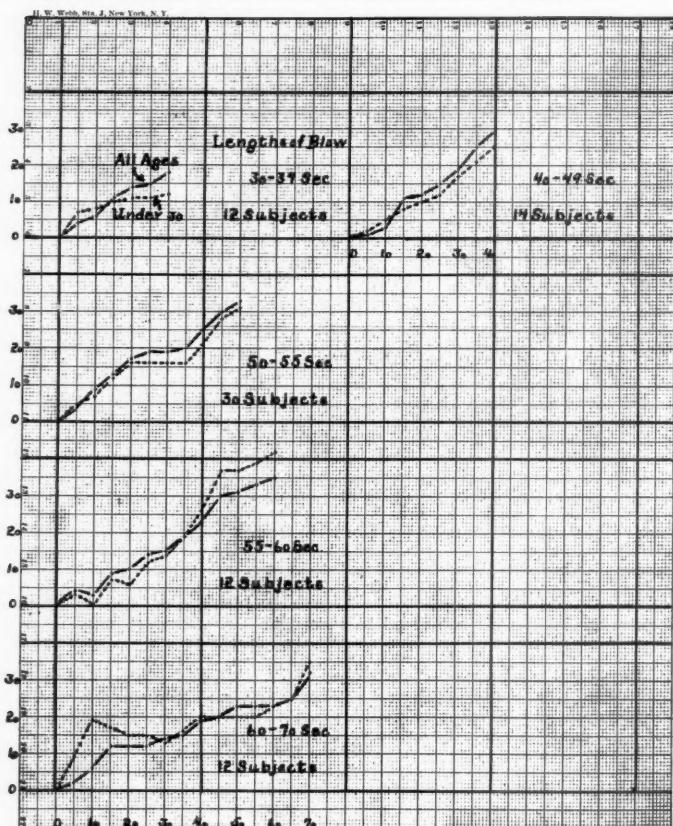
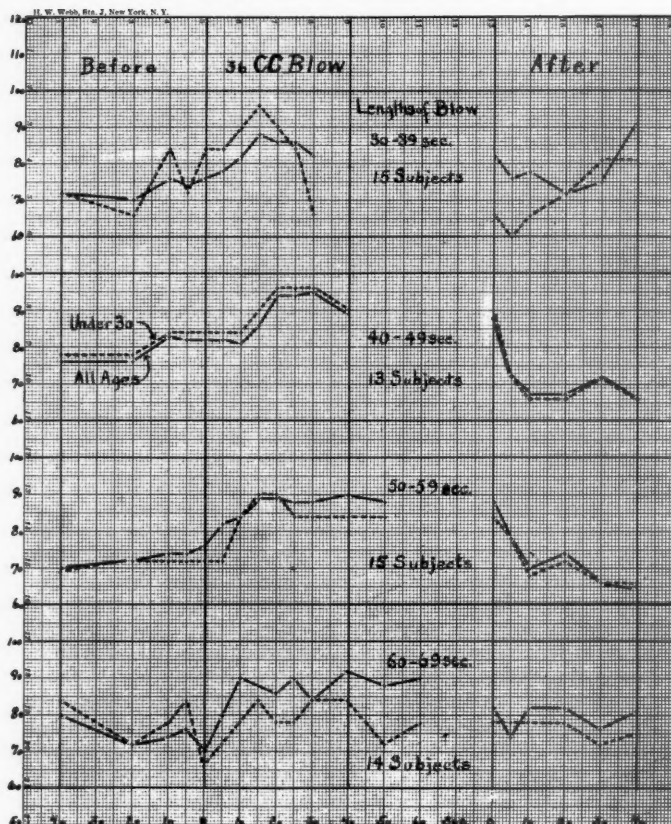




Fig. 7.
Pulse Rate in Flarimeter Tests



($S+$ is not greater than T_m , as shown in the last column), so that on this basis they should be regarded as normal.

Further experience may require changes in these limits, for excepting vital capacity they are based only on our very scanty data. Impaired applicants who desire insurance will stretch every nerve to lengthen the blow, which may lead to very different results from those on normal volunteers, mostly sedentary workers. These standards, therefore, are only preliminary and must await clinical and other evidence before their interpretation will have much weight.

The responses to the small orifice tests are so gradual that it is fairly easy to obtain complete time curves of an individual test, in fact all the tests in the Home Office were thus observed, to make sure that nothing was overlooked. But to combine these curves they must first be grouped by length of blow, or the reactions will be hopelessly confused. Thus grouped, the median curves for the eighty-eight normal males are shown in Figs. 5, 6 and 7, for the systolic and diastolic pressures and for the pulse rate. The medians for ages under 30 do not differ significantly from those for all ages, when grouped by length of blow.

The maximum systolic rise ($S+$) increases with lengths of blow over 40 seconds at a rate of about $1/3$ mm. per second, as shown by the lower right-hand curves in Fig. 5. Thus, the median rise is 10 less than the length of blow at 40 seconds, and about 30 less at 70 seconds, so that normal males do not give as large numbers for the systolic rise as for the length of blow. Nervously irritable subjects, however, do show a more rapid systolic response. Indeed, it is probable that the systolic curve for any subject will rise more and more rapidly if the blow is sufficiently prolonged. Yet general tendency is for the median responses to rise more slowly the longer the blow, so that at the end of the blow they have all arrived at about the same point. This indicates that subjects normally do not blow longer because of greater will power, but simply because it takes longer in them to produce the same distress.

A brief comparison has been made of the systolic pressure and heart rate responses of Royal Air Force and Flarimeter tests. The pressure and rate responses were followed simultaneously, first during the blow through the Flarimeter at 20mmHg., then during the blow against a closed mercury manometer at 40mm.Hg., each as long as possible after a full inspiration. Nineteen such comparisons on six subjects indicate that the responses are much alike. Usually, the systolic pressure does not rise quite as high, and sometimes the heart beats faster during the 40mm. blow, which averages 15 seconds shorter. It gives the subjective impression that increased muscular tension limits the length of blow and it is often stopped by fatigue of the lips.

Simultaneous observation of the systolic and diastolic responses while blowing through the small orifice of the Flarimeter has shown that the diastolic pressure rises while the systolic falls, then drops, and finally rises steadily until the end of the effort, but we have seldom observed any fall below the base-line, such as Dr. Amiral found to occur in the diastolic reaction to the large orifice blows of the cardio-respiratory test. In fact, the initial rise often merges with the final rise, making the initial diastolic rise even more difficult to observe than the initial systolic drop. The final drop in diastolic pressure below the base-line after the blow persists long enough to observe accurately, but unfortunately its magnitude is so small that it has little prospect of usefulness as a diagnostic criterion. The whole diastolic reaction is more difficult to follow than the systolic, and it is doubtful if the results would repay the extra effort, so that we have not attempted to include any diastolic responses in our routine technique.

The heart rate, on the other hand, can be counted continuously in five-second periods with ease. Simultaneous observations of heart rate and systolic pressure do not correlate as closely as do the systolic and diastolic, probably because of changes in the stroke volume of the heart. Henderson

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(1923) has shown that normally the stroke volume tends to remain constant, even when considerable increases in circulation are demanded as the result of exercise. In such cases the heart rate is a direct measure of the minute volume, and the product of heart rate and mean pressure is proportional to the mechanical power produced by the heart. But stroke volume must decrease during the initial systolic drop while venous return is impeded. The final responses in systolic, diastolic, and heart rate, on the other hand, may easily measure the power produced by the heart, for there is no such reason to expect changes in stroke volume at this stage of the blow.

Fig. 8 illustrates these points very clearly. The two upper curves are of systolic pressure, the middle diastolic, and the lowest heart rate, all on a single subject. Each curve is the average of three tests. The two dotted curves are simultaneous systolic (upper) and heart rate (lower); the two dot-and-dash, of simultaneous systolic (upper) and diastolic (lower). The two systolic average curves superpose very well after the first 25 seconds of the blow, in spite of the fact that they represent tests made six months apart, so that all the curves may be considered as if determined simultaneously. The final rises in systolic, diastolic and rate are remarkably parallel for 30 seconds, when the fall in heart rate signals the approach of the end of the blow. The systolic pressure, however, does not fall until the end of the blow, and then only momentarily, to be followed usually by a swift rise to its maximum some seconds later. The diastolic more nearly parallels the heart rate, but usually rises until the end of the blow and then collapses to values below the diastolic at rest.

In cases where the systolic, diastolic and rate increase together as the blow proceeds, it seems reasonable to assume that there is little change in stroke volume of the heart, and so the circulation through the brain is probably increasing steadily. Now according to Gesell (1925), the hyperpnea of

hemorrhage gives place to apnea simply by increasing the circulation through the respiratory center, without any changes in the blood. The apnea indicates that the acidity of the center is held down by the increasing flow of blood. If this is so during the small orifice test, the increased circulation would indicate that the respiratory stimuli are controlled by the will indirectly by means of involuntary vasoconstriction and heart acceleration. In spite of the accumulating CO_2 and vanishing oxygen in blood and tissues because of the apnea, the voluntary system has induced increased circulation through the respiratory center, and thus curbs its activity.

When the heart fails to accelerate, the stroke volume, to compensate, must steadily increase. Either process throws a progressive burden on the myocardium which can only be relieved by loss of voluntary control of apnea. This could be brought about by increasing respiratory stimuli until they become irresistible, or by anoxemia breaking down the voluntary control. Probably both are occurring at once. The respiratory center is known to be powerfully stimulated by anoxemia and excess CO_2 , and both these effects are here combined.

There is some indication that the stroke volume may increase at times during the later stages of the Flarimeter test. An example is given in Fig. 9, which, like Fig. 8, shows average curves of simultaneous systolic and diastolic (dot-and-dash, on left) and systolic and heart rate (dotted, on left). At the right are shown two of the individual tests, solid curves for one, the broken curves for the other. The bottom curve on the right is an average of several heart rate responses determined at the same period of time. All are on the same subject, but the dotted curves on the left represent tests made six months later than the others.

It is immediately evident that this subject is of a more nervous type than the one of Fig. 8. Moreover, the early systolic responses are more pronounced, exceeding 200mm.

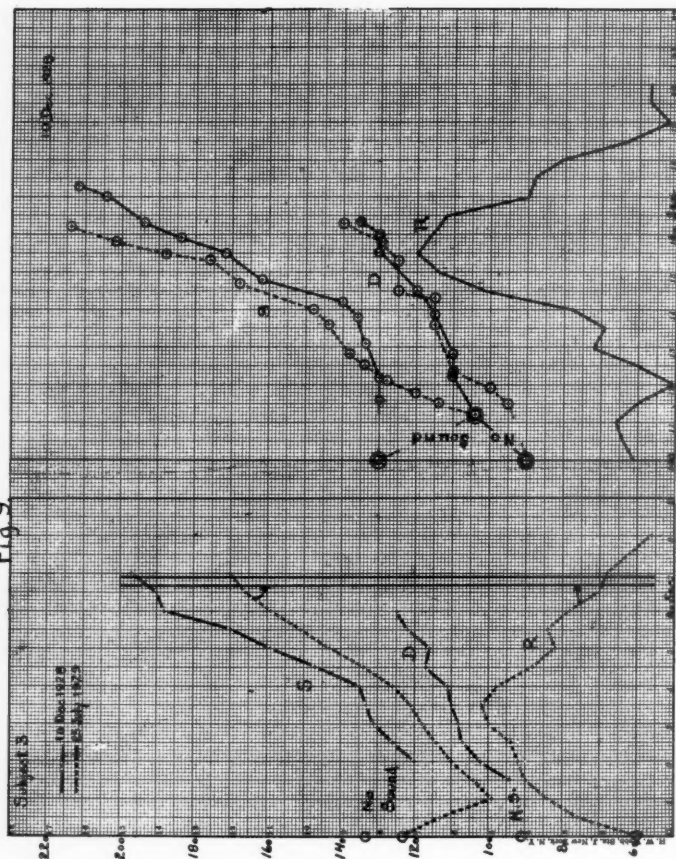
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Fig. 9



Hg. in individual tests. Six months later, the systolic at rest had fallen 10mm., and the maximum response from 63 to 44, without any appreciable change in the length of blow (69 sec. to 67 sec.). The pulse pressure increases throughout the blow, very slowly at first, then more rapidly as the heart accelerates, reaching values over 20mm. above its value at rest by the end of the blow. Six months later the heart rate rises only to 102 instead of to 120, and falls after the first 35 seconds. There is a corresponding effect on the systolic curve, but it continues to rise, nevertheless, at a rate which suggests increasing stroke volume, for vasoconstriction would hardly, by itself, produce such an effect in the face of decreasing heart rate.

That the subject of Fig. 9 is of nervous type is shown by the differences ($T_m - S +$), which vary from minus 22 to plus 16 in the early tests, and average plus 20 six months later. The values for Fig. 8 are 37 and 30 (plus). Yet subjects of phlegmatic type may push the blow far enough to force the systolic rise numerically above the length of blow, as shown by one of us who produced a 96mm. rise by blowing 84 seconds.

With the resumption of breathing there is normally a surge of blood through the heart until the venous pressure has had time to fall back to its rest level. But the vasodilatation which accompanies the marked slowing of the heart shows itself in the abrupt fall of diastolic as well as heart rate at the end of the blow to values below the base-line, and persisting until the circulation has reestablished normal conditions.

Our experience with functional tests indicates that the nervous fluctuations are the most serious of the disturbing factors. In the cardio-respiratory test, steps 2, 3 and 4 were repeated, and the median ranges of variation among the repeats are shown beside the median values in Tables 9 and 10. They are usually from one-third to one-half the response, comparing with ranges about one-half the final rise in steps

5 to 8. It is evident that the nervous factor is responsible for most of these variations. When Dr. Frost's reports are studied from this point of view it is apparent that he was fully aware of this source of error. He presented a chart to show "the tendency of excitement to exaggerate the response," and stated "We take pains to check up hyperactive reactions, whenever we consider it possible that excitement or apprehension have prevailed." But our results indicate that to trust the responses to the degree necessary to make detailed interpretations is unreliable, and to check up doubtful reactions is interminable. It is necessary to discount the hyperactivity of excitement and the hypoactivity of perfunctory performance.

Although T20 is the most reproducible of the small orifice responses, two out of the nine female athletes gave values less than 20 seconds. When the test was repeated on one of them, values of T20 ranging from 14 to 40 seconds were obtained. The reproducibility of T20 was therefore investigated in a normal male of age 21 and another of age 39. The boy showed the same nervous variability as the girl (age 19), but it was noticed that T20 tended to increase as the test was repeated, approaching a steady value after three or four tests (thus, one series was 14, 15, 27, 24, 36, 34, 34, 37, 36, 33, 36, 32 seconds). The 39-year-old male gave steady values from the start, the average deviation from the median T20 being only two seconds. It is, therefore, evident that the maximum values of T20 and the lengths of blow must be chosen as most characteristic. The longest blow is thus the best, and the systolic responses associated with it are the ones to be chosen.

To show that failure to inspire to full lung capacity is not the cause of these wide fluctuations in response, two male subjects were asked to perform the test without any preliminary inspiration, during tidal breathing, at "half-tide." Naturally, the subjects could not maintain the blow as long, which reduced T20 from 36 to 23 seconds in one (32 trials)

and from 44 to 32 seconds in the other (14 trials), with a difference in volume inspired of about 2 liters. This shows that ordinary fluctuations in full inspiration cannot account for differences of more than two or three seconds in T20.

There seems to be no quick method of eliminating the nervous factor. Certainly, one would expect T20 to be as free from such troubles as any criterion based on blood pressure responses. By standardizing the response at 20mm., variations in systolic level should be largely eliminated. But the fact remains that youthful subjects have shown considerable variability in this response, and the only method of handling it seems to be to repeat the test until the nervousness wears off. This is not the only field of measurement where selectivity is purchased at the price of stability.

CLINICAL CASES.

The following reports are on cases which were tested, at Beth Israel Hospital, with the Flarimeter and with each is the electrocardiogram and clinical diagnosis.

Prior to reviewing them we present some theoretical considerations and known facts upon which we base our attempts at interpretation of the reactions observed during the blow. A very large number of cases will have to be accumulated and considerable time will have to elapse before mortality ratios derived from the analysis of the responses will reveal their true significance.

During a blow against pressure resistance, preceded by a deep inspiration, a series of events occur which give rise to the reactions described.

There is first a marked rise in intrathoracic pressure which compresses the great veins and greatly reduces or completely shuts off blood supply to the right heart. Increased intrapulmonary pressure diminishes the caliber of the lung capillaries and this continues throughout the blow. In addition pressure is exerted on the heart, the right side being more affected. There is, therefore, some embarrassment of

the organ from interference with increase in size during diastole and possibly some impairment in systolic response as a result.

The right side, temporarily deprived of blood, cannot eject any into the pulmonary artery, circulation through the lungs drops and would completely cease if the condition continued. Because of this drop and because the blood in the lungs, at the beginning of the blow, owing to the increased intrapulmonary pressure is forced into the left chambers, these after being engorged for an instant are also practically emptied. Arterial pressure is, therefore, greatly reduced following a brief and sudden rise.

These changes are mechanical and if continued would very soon lead to circulatory standstill with collapse. This does not occur, however, unless the pressure applied is excessive or the individual so weak that a reasonable amount would be too great for him. Nothing approaching this condition has been observed with the Flarimeter.

While the changes mentioned have been taking place pressure in the cistern of Keith has been mounting and soon exceeds that in the thorax. This is due to the marked increase in intra-abdominal pressure, as not only is the diaphragm pushed down but the muscles of the abdominal walls are tense and retracted, thus reducing the size of the cavity. Vasomotor stimulation of the splanchnic veins is also a factor. A squeezing effect results which must be more than sufficient to overcome the pressure on the abdominal portion of the inferior vena cava, the right heart is again filled, pulmonary circulation increases, the left heart fills rapidly, and arterial pressure promptly rises. Venous pressure does not return to its level before the blow but increases, as it must continue to overcome increasing intrathoracic pressure.

At the time of fall in the arterial pressure coronary and brain circulation are reduced, stimulating vasoconstriction of the systemic arterioles and the diastolic pressure rises. As soon as systolic pressure is restored, peripheral constrict-

tion becomes less intense and the diastolic may drop slightly but quickly begins to rise with the systolic and continues in that relation to the end of the blow.

MacLeod (p. 487) says Evans and Starling found that "Changes in coronary blood flow depend intimately on changes in aortic blood pressure; for example, an increase of 50 per cent. in aortic pressure may cause the coronary flow to increase three times." He further says, "It is computed that the blood flow through the heart of a man during rest is 140 cc. per minute; during muscular exercise the flow may increase to 800 cc. Under these conditions the oxygen consumption of the heart is increased in greater proportion than the increase in blood flow, which indicates that the O_2 must be more thoroughly utilized, i. e., the coefficient of utilization becomes greater."

While, as has been pointed out, the heart is not under the same kind of strain in this test, the fact remains that increased activity does occur and this in the face of blood poor in oxygen.

Just before the blow is started as deep an inspiration is taken as possible. This increases the volume of fresh air in the lungs, but about one-eighth of it will remain in the trachea, bronchi and bronchioles (dead space) so that the air in the alveoli is less than one-third fresh. According to Haldane and Henderson the dead space is much larger, which would reduce the fresh air to less than one-fifth.

Motion in the lungs is almost at a standstill with greatly reduced mixing of gases. Diffusion is slow and the capillaries have to depend for oxygen supply on the layer of oxygen in the air at the alveolar wall. This amount must decrease, reducing the oxygen supply as the blow continues. Aeration of the blood is becoming continuously poorer and escape of CO_2 interfered with for the same reason.

The pressure of oxygen in the alveolar air falls more than 40 mm. when the breath is held for forty seconds (Douglas & Haldane, Meakins & Davies). The latter investigators

even found a fall in the oxygen saturation of the haemoglobin in the radial artery of over 13 per cent.

The time required for pulmonary circulation is about 15 and for the systemic about 60 to 75 seconds. Coronary circulation is more rapid than in any other part of the body. The pulmonary circuit will have been completed from about two to five times and the coronary many times during the blow, the systemic not more than once. Few blows are under 30 or over 75 seconds in length, the average being about 50.

As the blood pressure rises more and more blood is being deflected from the aorta into the coronaries and its oxygen more rapidly extracted. This blood, unlike that of the systemic, is immediately thrown back into the pulmonary circuit where raising its oxygen value is becoming progressively more difficult, even if there is active oxygen secretion. A vicious circle occurs which results in continued lowering of oxygen content in both coronary and systemic arteries, but especially the latter.

That increased work is demanded of the heart is evidenced by the rising blood pressure. The rising metabolic rate requires an ever increasing oxygen consumption and this can lead to but one of two results, either an irresistible stimulus to stop the blow or severe myocardial fatigue.

We have pointed out that there is a definite and increasing lack of oxygen in the systemic circulation as a result of the blow with an increasing CO_2 content. While the latter stimulates the vasomotor, its chief influence is exerted on the respiratory center. Oxygen lack will not be tolerated by the brain and when present gives rise through the vasomotor center to constriction of the peripheral arterioles with rise in diastolic pressure. Constrictor action is weak in the brain vessels, blood is diverted from the tissues of the body to this organ and flow through it is increased in an effort to compensate for lowered oxygen. To meet this, pulse pressure must be maintained and the systolic also rises.

Individuals vary in the inherent tone and resisting power (will power) of their cortical centers and this plays a prominent part in the length of time taken for the blow. The functional ability displayed is nevertheless largely a matter of oxygen supply. Increased expenditure of energy of these centers, in an effort to prolong the blow, combined with oxygen shortage produces fatigue of the centers with loss of inhibitory control of the basal nuclei. On the other hand, emotional stimuli may so affect the lower centers that exaggerated reactions and shortened blows result. It has already been indicated that the respiratory center is controlled, within individual limitations, by the cortical centers. Rising carbon dioxide and falling oxygen content both stimulate this center, the CO_2 effect being the more powerful.

Deficient oxygen impairs all the centers, while excess carbon dioxide excites the respiratory and this center sooner or later dominates. It is obvious, therefore, that the nervous responses to the Flarimeter test are characteristic of changes taking place in the circulatory system and so intimately involves the heart. If such a load can be thrown upon the normal heart, what must be the effect on a myocardium already impaired?

Professor F. R. Fraser (1927) has summarized his masterly Goulstonian Lectures on cardiac dyspnea as follows:

"SUMMARY AND CONCLUSIONS.

"1. The study of the arterial blood in cases of cardiac dyspnea shows the presence of factors producing increased ventilation or hyperpnea, and indicates that: (1) The constant stimulus to the respiratory center in cardiac dyspnea to produce increased ventilation is a deficient supply of oxygen, due to a diminished blood-supply to the center. (2) In cases with pulmonary disease in addition, and cases of severe failure with pronounced secondary changes in the lungs, raised carbon dioxide pressure and increased hydrogen-ion concentration will act as an additional stimulus. (3) In cases near death, or in very advanced conditions of failure, the retention of non-volatile acid substance will result in

an increased hydrogen-ion concentration that will act as an additional stimulus. (4) In some cases under (2) and (3) a low oxygen saturation of the arterial blood may be present, and this will be a further factor in the stimulation of the center.

"2. There is evidence that the slow circulation rate of heart failure which results in the stimulus at the center to produce increased ventilation, results at the same time in reduction of vital capacity by engorgement of the pulmonary circulation and in fatigue of the center, so that the need for increased ventilation cannot be met without consciousness of the respiratory movements. This produces the distress of dyspnea.

"3. The clinical features and experimental observations on orthopnea, periodic breathing, and paroxysmal dyspnea (or cardiac asthma) are compatible with these conceptions of cardiac dyspnea.

"4. Dyspnea may be a dangerous reaction, and in certain cases the direct treatment of it by means of morphia is indicated, but full use of this action of morphia cannot be attained until practical methods of administering oxygen are further developed.

"5. The fundamental cause of cardiac dyspnea is inefficiency of the myocardium."

Blood changes in anemia, diabetes and nephritis not only impair cardiac tone but add to its burden as all parts of the body also suffer and demand increase in supply to meet as far as may be decrease in quality. Toxic goiter, as is well known, damages the myocardium. Overweight through infiltration of and deposits on the tissues and organs interferes with their circulation and function, while in the abdominal area it may cause ptosis and pooling of blood. The heart itself is embarrassed by such deposits both on and within it. In all these conditions then the myocardium is directly impaired by deficiency in oxygen, by toxic conditions (acidosis), by increased rate with hypertrophy (goiter) and by embarrassment from excess fat in the tissues. Everything points to the value of the Flarimeter as a test of disturbance of functional capacity and of quality of blood in the abnormalities above cited.

The test having the longest blow is the one we have used in the interpretation, though the records of the one preceding or following may often influence the conclusion.

Before presenting the thirteen cases described on the forms which follow and to each of which is attached the electrocardiographic tracings we will describe the abbreviations used.

TENTATIVE LIMITS FOR INTERPRETATION.

Variable	Symbol	Pathological	Subnormal	Normal
Vital Capacity	V	under 85%	85-90%	90% and over
Initial drop in systolic during blow	S-	under 5mm.	5-15mm.	15mm. and over
Final Rise to maximum systolic at end of blow	S+	under 20mm.	20-30mm.	30-50mm.
Time for systolic to reach 20mm. above that before beginning blow	T20	under 25 sec.	25-35 sec.	35 sec. and over
Time taken to complete blow	Tm	under 40 sec.	40-50 sec.	45 sec. and over

The word "subnormal" is used to signify a reaction lying between the normal and the pathological borderlines indicated in the table. The Vital Capacities over age forty have all been corrected, the reasons for this having already been given.

It is most unfortunate that Dr. Jacob Polevski, attending physician of the Beth Israel Hospital, who manifested great interest in the tests and cooperated so splendidly with us, was taken sick and so was not able to interpret all of the electrocardiograms. Nor are those to which his name is attached as complete as we believe he would have liked. But the electrocardiographic tracing is attached to each case, and so anyone will have an opportunity to interpret them.

There is no doubt that the tests give a much better idea of blood pressures prevailing in the ordinary life of the applicant than can be obtained from single readings of the systolic, diastolic and pulse. It is premature to enter into detailed interpretation of the responses until more clinical material is available for classification. Only two are yet of clear-cut significance—vital capacity and breath-holding time. With regard to the others some queries and suggestions may

not be out of place. Two abnormal types may differentiate themselves, one type, the hypertensive, in which the blood pressure rises easily but does not fall much—another type, the hypotensive, in which the pressure falls but does not readily rise. It looks as if a normal circulation should not react too soon to the increased intrathoracic pressure, and so the systolic drop should not be too small. One wonders if there should also be an upper pathological borderline for S—, or is venous pooling and lowered tone sufficiently indicated by a small systolic rise (S+) and shortened blow (Tm)?

We are just beginning to see the factors involved in T20, and so far have no reason to set an upper limit as abnormal. If 20mm. is the pathological border line for the systolic rise (S+), reaching T20 in itself would be a normal sign. Short T20's might be an indication that the vasomotor system is compensating for an impaired heart. On the other hand, the nervous factor may upset and reduce this criterion to a mere sign of irritability. The heart rate may give valuable indications when its behavior during this test is better known.

CONCLUSION.

Gentlemen: We began our investigations of cardio-respiratory tests because the many reports, by Dr. Frost to this Association, suggested that the one he had designed was of real value in medical selection. Two of us have at different times visited the Medical Department of the New England Mutual and wish to express our appreciation of the cordiality with which we were always received by Dr. Dwight, Dr. Frost and the other members of their departments. We spent one day with them discussing their test and being taught by Dr. Frost to perform it.

As a result of our researches we have modified the apparatus, made changes in technique and have placed great emphasis on breath-holding time as the best criterion. We have also reduced to a minimum the applicant's difficulties, as all we require of him is to learn how to take a deep in-

spiration and to hold the water at a fixed level during a continuous blow.

Now gentlemen, length of time taken for application of any test during examination for life insurance is a very important matter indeed and we have done our best to reduce it to a minimum and yet obtain information consonant with improvement in medical selection. We all remember Dr. Rogers' comment, when discussing Dr. Frost's last paper, that "The groups have been blocked out as a whole. It now remains for us to find a means of distinguishing within the groups between those who are better than their class and those who are less desirable." Our researches, we believe, give much promise in this direction. We do not think that the test is advisable in all cases, but certainly feel that it will prove of real value in cardiac impairment per se, and also in many others which may have caused heart muscle change not detectable by ordinary methods. As examples we mention albuminurics and overweights. Surely in applicants who appear impaired we are fully justified in prolonging an examination a few minutes if it seems that better selection will result. Again, any applicants who really want insurance, and most do if they think or know they are impaired, would not object to the time element, would put forth the greatest effort of which they are capable, hoping thereby to make an excellent showing, and would in this way materially improve the value of the test. Simplicity then can be obtained only at the expense of desirable and important information.

The time requisite for performance in the field is about fifteen minutes, but in reality it is less than that, for blood pressure and pulse rate at the time of examination may be used for test one, or if the test is applied first, then the data necessary for completion of the report could be used. The exercise test would be used in a similar manner so that the additional time taken would be only that necessary to determine vital capacity, to make three small orifice blows and to

take final pulse rate and blood pressure readings; when properly done these take no longer than nine minutes. Of course, skill of the physician in applying the test and intelligence of the applicant in learning to blow properly really determine the time consumed. Our doctors have had little trouble in learning to do the test and with rare exceptions, in either Field or Home Office, has difficulty been experienced in having the blow done properly by an applicant. We believe that "any man with fair training anywhere" can learn to perform this test, especially if he is personally taught.

We have kept Dr. Frost's pressure standard (20mm. Hg.) and have included his orifice so that his test may be applied with the Flarimeter, except for the 40mm.Hg. pressure blow and Step 3. This step we believe, from correspondence with him, he does not value highly because of faulty performance.

We have combined exercise with our test because its effect is different, as already explained, and therefore tests heart function in quite another way. The degree of dyspnea following the exercise may be determined by having the applicant sit down immediately following the test and at once blow into the Flarimeter as long as possible. The blow will be very much shortened and this shortening in comparison with the longest previous blow may prove to be of definite significance.

In any test it is not desirable to have more than one variable at a time and for that reason we have standardized on a single intrapulmonary pressure. This permits including all (S—, S+, T20, Tm) or discarding, one, two or three of them, and still have results perfectly comparable with similar ones of other companies. In fact, the series of tests used permit exclusion of one or more procedures without impairing the comparability of the others. This is a real advantage as some may not care to adopt the test in toto, but the portions they do use will always be of service if a number of companies should wish to combine in investigation of

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Synopsis of Technique for Functional Tests

Test I	Tests III, IV.	Test V	Test VIII
Pulse Rate	Seconds	Seconds	Seconds
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60 Standard Exercise
Diastolic Pressure	Deflate Record	Record Remove Cuff	60-50)
(4th Point)	55-60 Inflate " Inspire! "		60 " Blow! "
	60 " Blow! "	Tests VI, VII	60-60) Heart Rate (per 5 sec.)
Test II	60-20 Systolic Drop to Minimum	30-55 Heart Rate (per 5 sec.)	60-60) Heart Rate (per 5 sec.)
	Deflate Record	Record every 5 seconds	Record every 5 seconds
Vital Capacity	25-15 Inflate— T20 in seconds	60 " Blow! "	Underline End of Blow
Seconds X 0.2 = Liters	Maximum Systolic	60-30) Heart Rate (per 5 sec.)	
<i>Repeat</i>	Total Length of Blow	Record every 5 seconds	Continue counting till original rate is reached
	<i>Repeat</i>	Underline End of Blow	
		<i>Repeat</i>	

RECORD OF TESTS

Name..... Age..... Sex..... Occupation..... Ht..... R..... Wt..... Ins.....

I	III	IV	V	VI		VII	VIII
At Rest	Systolic Response-Fluorimeter		At Rest	Heart Rate Response-Fluorimeter		After	Number Ascents
Pulse Rate				Seconds	Before	During and After Blow	During and After Blow
				0-5			
				-10			
Systolic	Systolic		Systolic	-15			
	Minimum Systolic		Diastolic	-20			
Diastolic	T20			-25			
	Seconds			-30			
Vital Capacity	Maximum Systolic			-35			
Sec. Liters				-40			
1	Total			-45			
	Seconds			-50			
2				-55			
These lines for Home Office use only				-60			
% of Normal/Vital Capacity	S—			Length of Blow		Sec.	Sec.
	S+						

Remarks:

HOME OFFICE

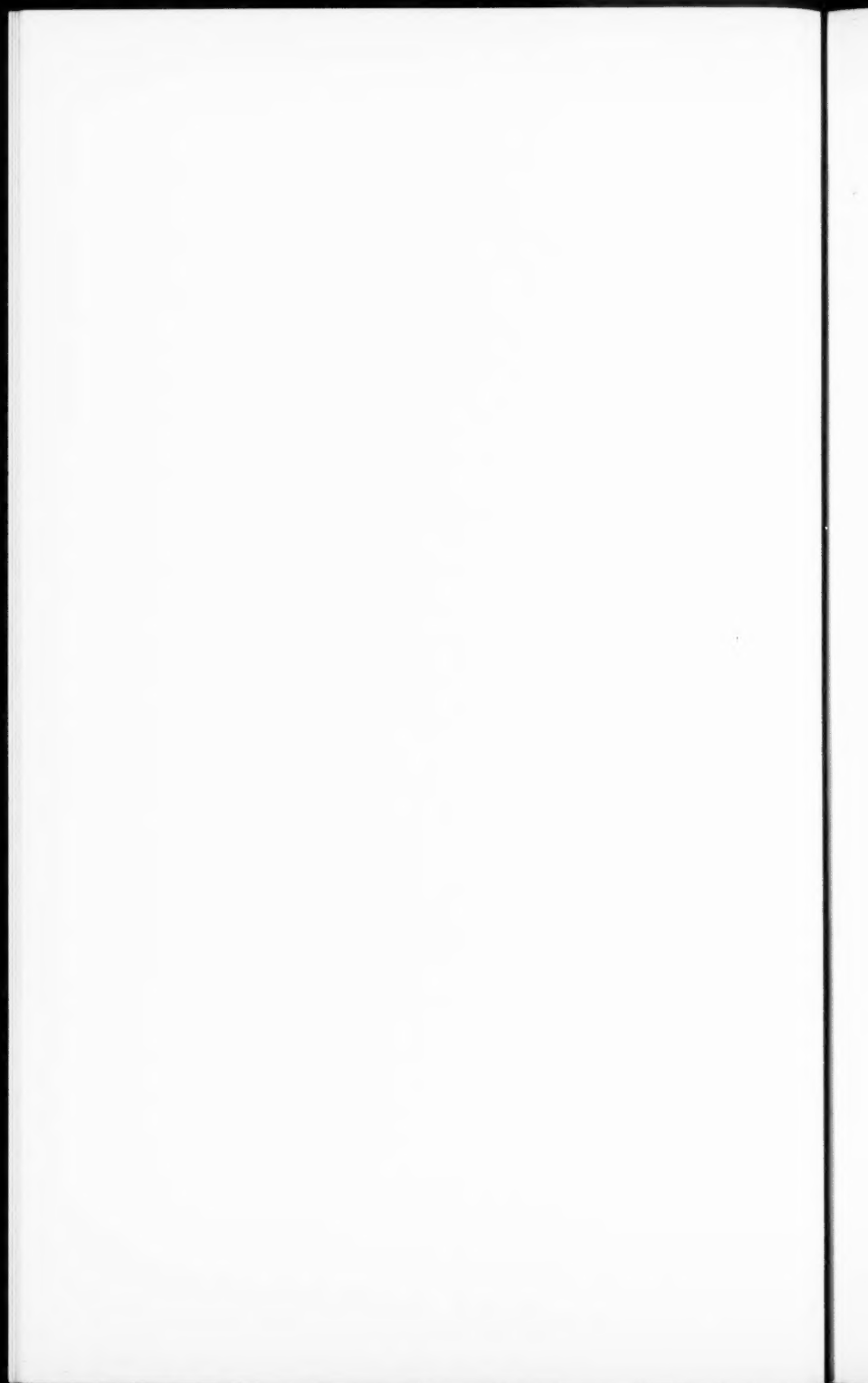
Examiner..... Residence..... Date..... Hour.....

Synopsis of Technique for Functional Tests

RECORDS OF TESTS

Remarks:

Examiner	Residence	Date
84410		



the significance either of a particular test or any point in the systolic curve derived from the blow.

You all know the objection to tests of this type is that but one observation is made and that a person's tone varies so from hour to hour and day to day that its application may lead to faulty conclusions. It must be frankly admitted that there is reason in such an argument. We are, however, continually drawing conclusions from urinalysis and blood pressure readings which will give even more unreliable information, as neither are as searching in character but will vary as much and probably more. We have the same privilege with this test as we have with the others, of asking for another. The slight increase in expense involved can hardly be advanced as a valid argument if the test fulfills its present promise.

Some possibly feel that the clinical cases presented would all have been rejected anyway with very cursory examination and that sufficient selectivity is far from being established by the records shown. We admit this frankly, but hope that we have already given enough reasons for expecting results. Furthermore, we have confirmatory evidence from cases so far reviewed. Unfortunately the data we have been able to accumulate is very small, as use of the instrument in the field has only begun. This being a preliminary report, we will by the time another year has elapsed be able to speak with more certainty as to its actual value.

Appended are two forms—one for use in the field, the other, more elaborate, we are using in our Home Office researches. Time and accumulation of data will alone show what alterations in the form will be advisable and how simple it can be made without seriously decreasing its value. At present we would not care to change the one now being used in the field. With the forms will be found "Directions" which apply more particularly to our Home Office form, but also to use of the Flarimeter with the field form, except that

the heart rate responses and the initial systolic drop are omitted. Comparison of the forms will quickly indicate the differences.

It may be that some of the Companies represented would like to join us in our investigation of the relative merits of systolic pressure and heart rate. If they do, we will be glad to furnish them with copies of our Home Office form and will appreciate receiving duplicates of records made by them; due credit, of course, being given each Company so cooperating. Definite information could be obtained at a much earlier date if a sufficient number would unite with us in this way.

Before closing, we wish to acknowledge our obligation to Dr. M. J. Kaufman, who is one of our examiners in Newark and an attending physician at Beth Israel Hospital, for his great interest and help in securing clinical material and obtaining the records previously reported; also to the members of our office force who so willingly performed all tests they were asked to undergo.

Synopsis of Technique for Functional Tests

Test 1

Pulse Rate
Systolic Pressure
Diastolic Pressure
(4th Point)

Test II

Vital Capacity
Seconds $\times 0.2$ = Liters
Repeat

Tests III, IV

Seconds
25-45 **Systolic Pressure**
Deflate Record
55-60 Inflate "**Inspire!**"
60 "**Blow!**"
60-20 Systolic Drop to **Minimum**
Deflate Record
25-15 Inflate—**T20** in seconds
Maximum Systolic
Total Length of Blow

Repeat

Test V

Seconds	
30-60	Systolic and Diastolic
Record	Remove Cuff

Tests VI, VII

30-55 **Heart Rate** (per 5 sec.)
Record every 5 seconds
60 "Blow!"
60-60 } **Heart Rate** (per 5 sec.)
60-30 } Record every 5 seconds
Underline End of Blow
Repeat

Repeat

Test VIII

Seconds
 20-60 } **Standard Exercise**
 60-60 } **60 "Blow!"**
 60-60 } **Heart Rate (per 5 sec.)**
 60-60 } **Record every 5 seconds**
 Underline End of Blow

 Continue counting till original
 rate is reached

RECORD OF TESTS

Name C. B., C. 3 Age 56 Sex M Occupation Jeweler Ht. 5 ft. 5 ins. Wt. 210 lbs.

I		III		IV		V		VI				VII		VIII	
At Rest		Systolic Response-Fluorimeter				At Rest		Heart Rate Response - Fluorimeter				After Number		Ancients	
								Seconds: Before		During and After Blow		During and After Blow		During and After Blow	
Pulse Rate 65								0-3		5 5		5 5			
								-10		6 6		5 5			
Systolic 146		Systolic 136				Systolic 140		-13		6 5		5 5			
		Minimum Systolic = 130				Diastolic =		-20		7 -		5 5			
Diastolic 68								-25		9 6		5 5			
II		Seconds 35 30						-30		7 -		7 5			
Vital Capacity 184		Maximum Systolic 184				180		-33		8 5		7 4			
		Total Seconds 45 43						-40		6 5		8 -			
1 17 3,4								-43		7 5		8 5			
2 18 3,6								-50		6 5		5 5			
								-55		6 5		5 5			
								-60		5 5		5 5			
% of Normal Vital Capacity 105		S + - 10						Length of Blow 40		Sec 45		Sec		Sec	
		S - 46 40													

Remarks: Clinical diagnosis- myocardial degeneration.

g. E_g- Myocardial changes.

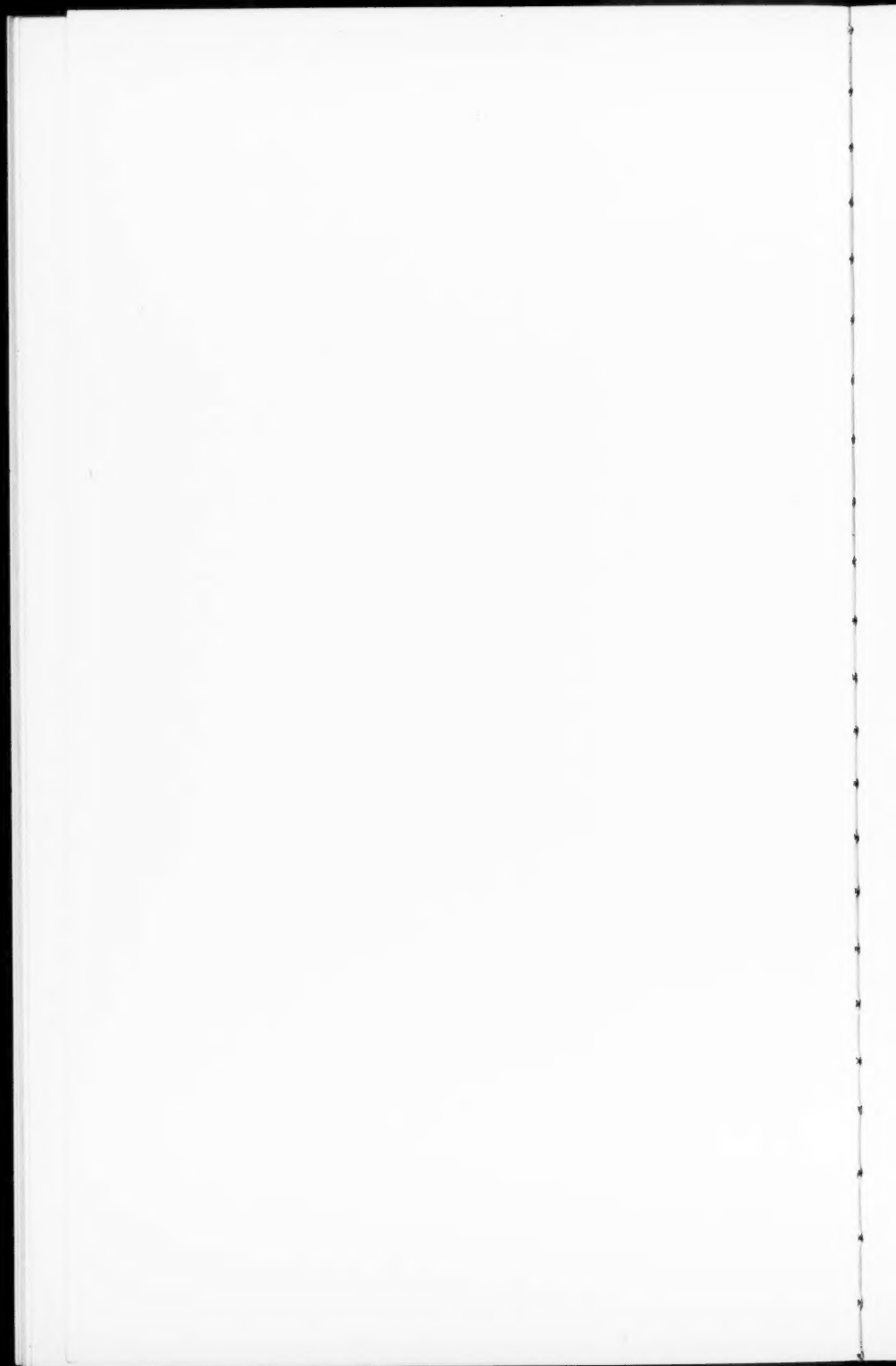
Dr. Polevaki.

Flarimeter- V.C. as corrected is high. S- Missed the first, but subnormal the second blow. S $\frac{1}{2}$ -Normal. T20 Subnormal. Tn Subnormal.

Pulse rate, systolic and diastolic blood pressures, and vital capacity suggest rather better results than shown by the blows, but the latter are more in keeping with the applicant's real condition which appeared good except for overweight and somewhat florid face.

Examiner Residence Date 7/23/29 Hour 9.15 P.M.
98405

9560



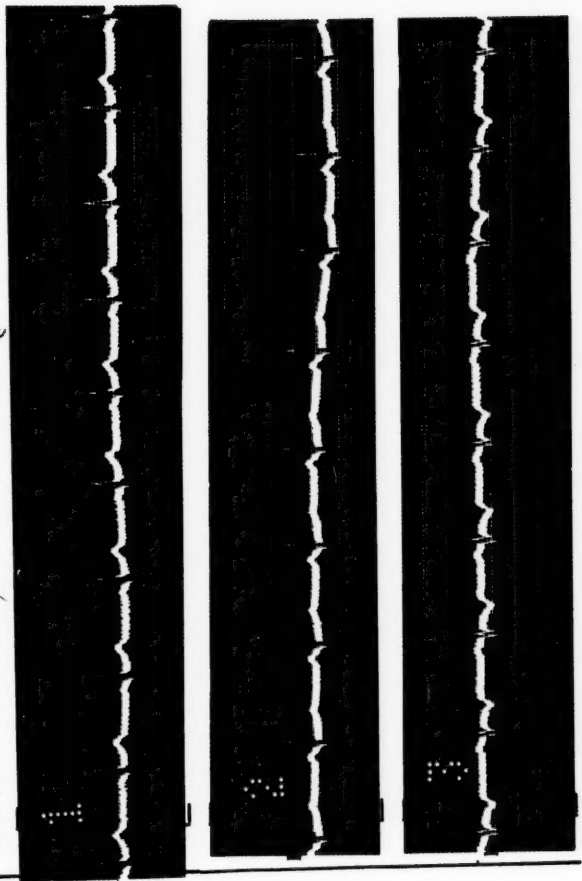
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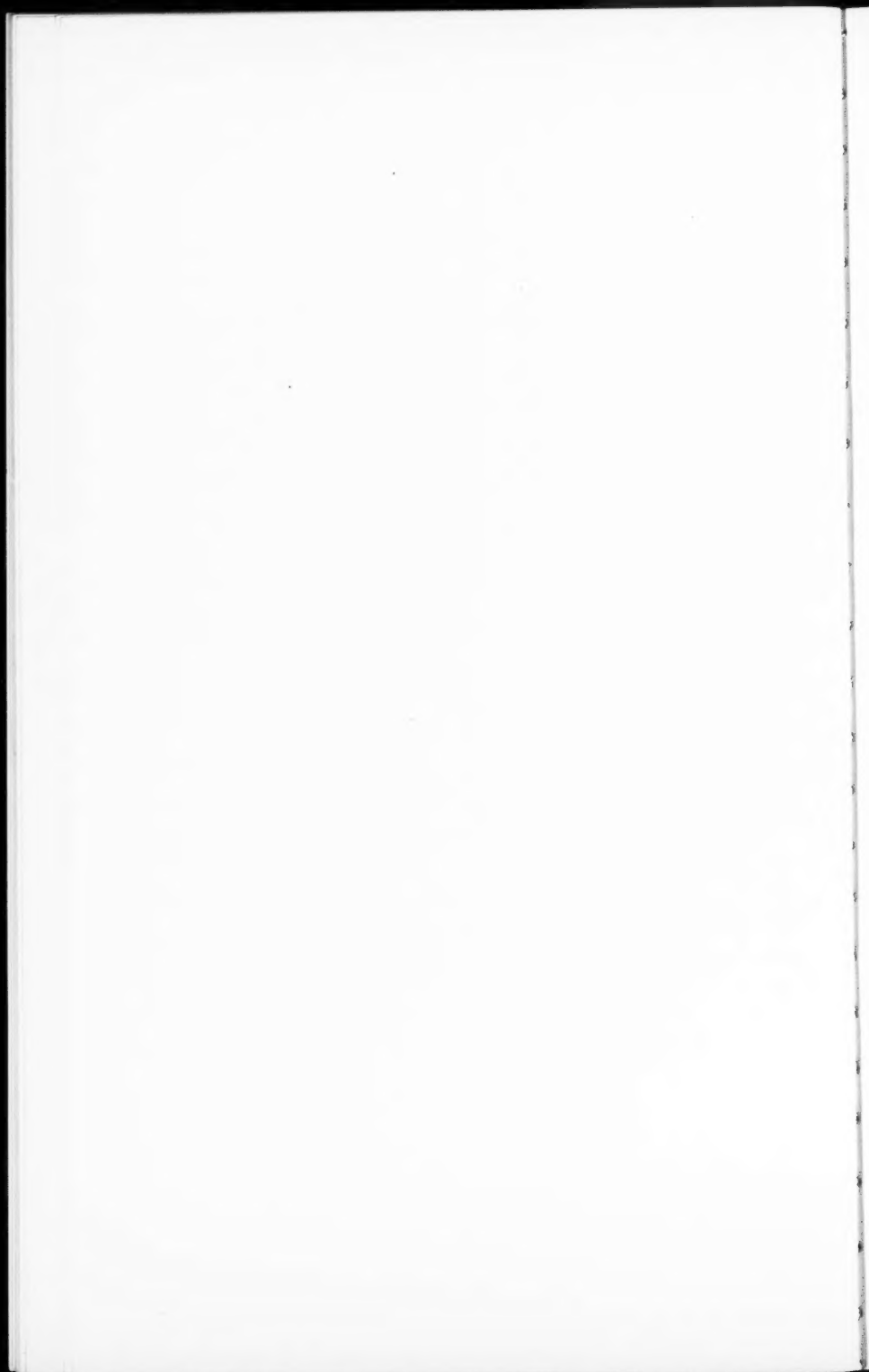
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Synopsis of Technique for Functional Tests

Test I Pulse Rate Systolic Pressure Diastolic Pressure (4th Point)	Tests III, IV Seconds 25-45 Systolic Pressure Deflate Record 55-60 Inflate "Inspire!" 60 "Blow!" 60-20 Systolic Drop to Minimum Deflate Record 25-15 Inflate—T ₂₀ in seconds Maximum Systolic Total Length of Blow	Test V Seconds 30-60 Systolic and Diastolic Record Remove Cuff Tests VI, VII 30-55 Heart Rate (per 5 sec.) Record every 5 seconds 60 "Blow!" 60-60 Heart Rate (per 5 sec.) Record every 5 seconds Underline End of Blow Repeti	Test VIII Seconds 20-60 Standard Exercise 60-50 "Blow!" 60-60 Heart Rate (per 5 sec.) Record every 5 seconds Underline End of Blow Continue counting till original rate is reached
Test II Vital Capacity Seconds X 0.2 = Liters Repeti	Repeti	Repeti	Repeti

RECORD OF TESTS

Name F.B., C. I. Age 46 Sex M Occupation Delivery Truck Ht. 5 ft. 2 in. Wt. 118 lbs.

I		III IV		V	VI		VII	VIII
At Rest		Systolic Response- Flarimeter		At Rest	Heart Rate Response-Flarimeter			Number After Anasuta
					Seconds Before	During and After Blow	During and After Blow	During and After Blow
Pulse Rate	80				0-5			
Systolic	122	Systolic	130 122	Systolic	-10			
Diastolic	70	Minimum Systolic	112 96	Diastolic	-15			
		T ₂₀ Seconds	28 =		-20			
		Maximum Systolic	166 130		-25			
Vital Capacity Sec.	16.5 3.3	Total Seconds	98 55		-30	Not made		
					-35			
					-40			
					-45			
					-50			
					-55			
					-60			
					-65			
					-70			
					-75			
					-80			
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Remarks: Clinical Diagnosis- Aortic-stenosis- mitral regurgitation.

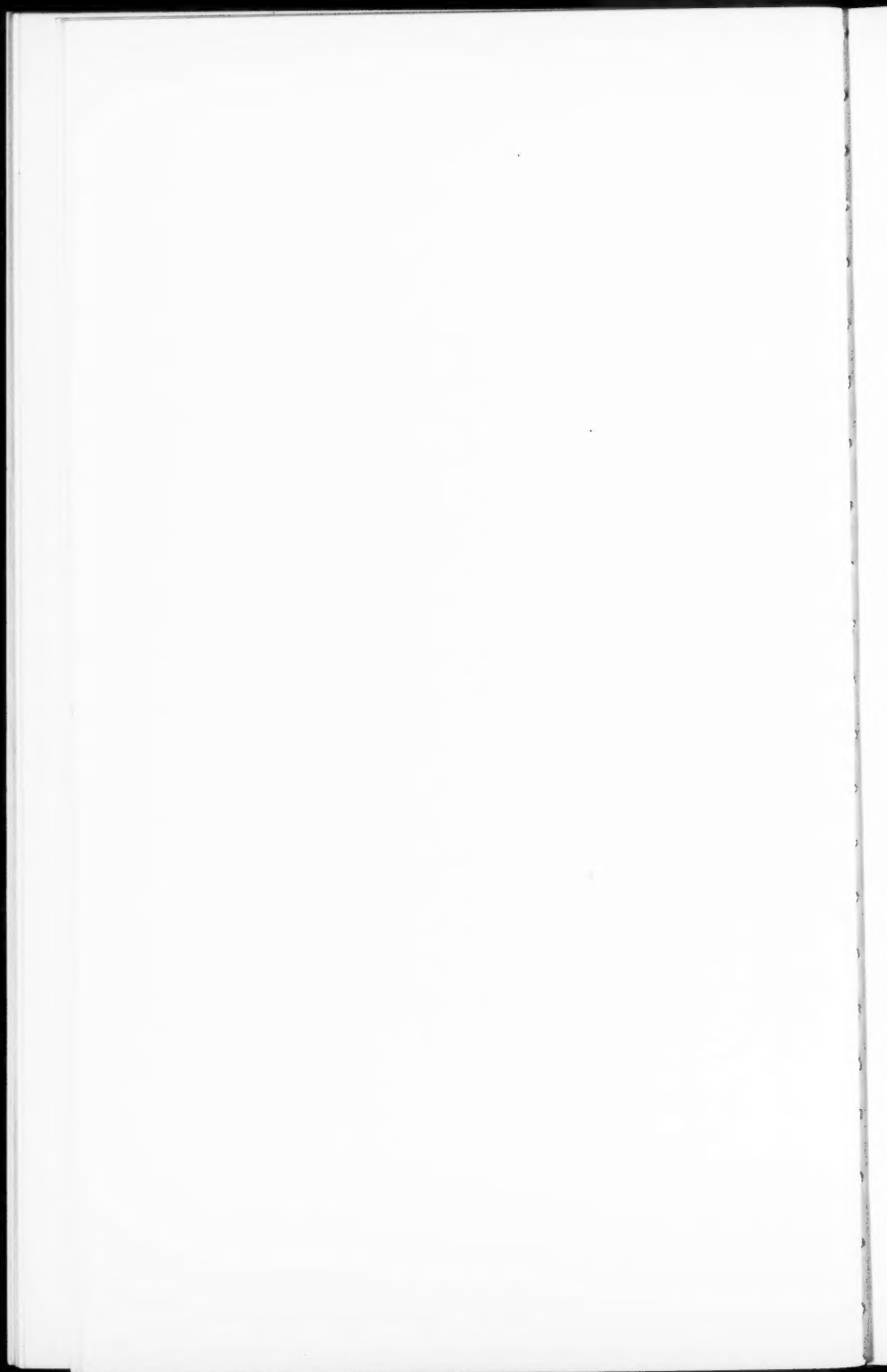
Chest 27.2 cm., Left heart 7.2, Rt. heart 5.0. Ratio heart to chest 45%.

E.E.- Myocardial degeneration. Inverted T in all these.

Flarimeter- V. C. Normal. S- marked. S+ Subnormal, second blow markedly pathological. T₂₀ Subnormal; taken with the second blow, however, indicates a pathological condition. T₂₅ Normal.

All the findings suggest myocardial impairment with irritability. The test does not, however, point to as serious a condition as is revealed by electrocardiogram, although the second blow does show much greater impairment than the first.

Examiner.....Residence.....Date 7/23/29 Hour 8.45 p.m.
99605



Synopsis of Technique for Functional Tests

Test I	Tests III, IV	Test V	Test VIII
Pulse Rate	Seconds	Seconds	Seconds
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60 Standard Exercise
Diastolic Pressure	Deflate Record	Record Remove Cuff	60-30
(4th Point)	55-60 Inflation "Inspire!"		60 "Blow!"
	60 "Blow!"	Tests VI, VII	60-60 Heart Rate (per 5 sec.)
	60-20 Systolic Drop to Minimum	30-55 Heart Rate (per 5 sec.)	60-60 Heart Rate (per 5 sec.)
	Deflate Record	Record every 5 seconds	Record every 5 seconds
Test II	25-15 Inflation—T20 in seconds	60 "Blow!"	Underline End of Blow
	Maximum Systolic	60-60 Heart Rate (per 5 sec.)	
Vital Capacity	Total Length of Blow	Record every 5 seconds	Continue counting till original rate is reached
Seconds $\times 0.2 =$ Liters		Underline End of Blow	
Repeat	Repeat	Repeat	

RECORD OF TESTS

Name **M. B. Q. 4812** Age **75** Sex **M** Occupation **...** Ht. **5** Wt. **160**

I	III	IV	V	VI	VII	VIII
At Rest	Systolic Response-Flameter		At Rest	Heart Rate Response-Flameter		Number Ascents
Seconds	Before	During and After Blow	During and After Blow	During and After Blow		
Pulse Rate 68				0-5		
Systolic 155	Systolic	158 154	Systolic	-10	5 5 7 8	
Diastolic 78	Minimum Systolic	130 -	Diastolic	-15	5 5 5 7	
	T20 Seconds	- -		-20	5 5 5 7	
	Maximum Systolic	135 172		-25	5 5 5 5	
Vital Capacity	Total Seconds	27 25		-30	5 5 5 5	
Sec. Liters				-35	5 5 5 5	
1 17 3.4				-40	5 5 5 5	
2 15 3.0				-45	5 5 5 5	
				-50	5 5 5 5	
				-55	5 5 5 5	
				-60	5 5 5 5	
% of Normal Vital Capacity 111	S- 28	-23 18		Length of Blow	35	Sec. 34
	S+ -23 18					

Remarks: Tests III, IV and VI, VII were not made at same time.
Ward case confined to bed.

Width chest 31.5 cm., Left heart 12.5, Rt. heart 6.5, Ratio heart to chest 60%.

E. K. shows distinct myocardial degeneration. Right bundle block not excluded.

Dr. Polevaki.

History of rheumatism 15 years ago.

Flameter-V.C. Corrected high. Correction in this case probably too great, as the man is seventy-five years of age. Uncorrected percentage 78, which is pathological. S- Marked. S+ Not only pathological but actually minus 23. T20 absent.

Tn Pathological.

Test showed without a doubt that a very serious condition existed.

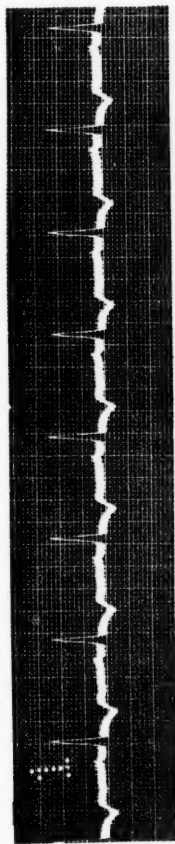
Examiner **...** Residence **...** Date **7/18/29** Hour **10:30 a.m.**



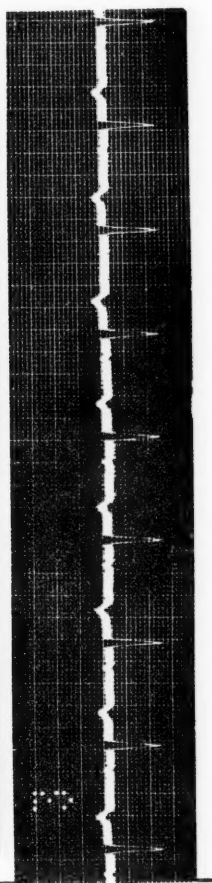
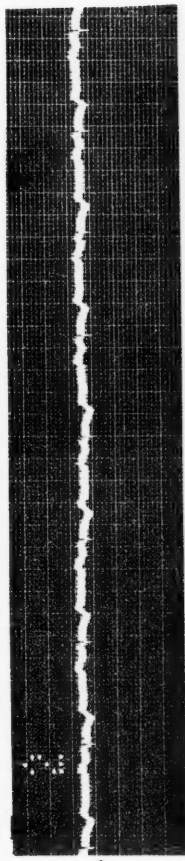
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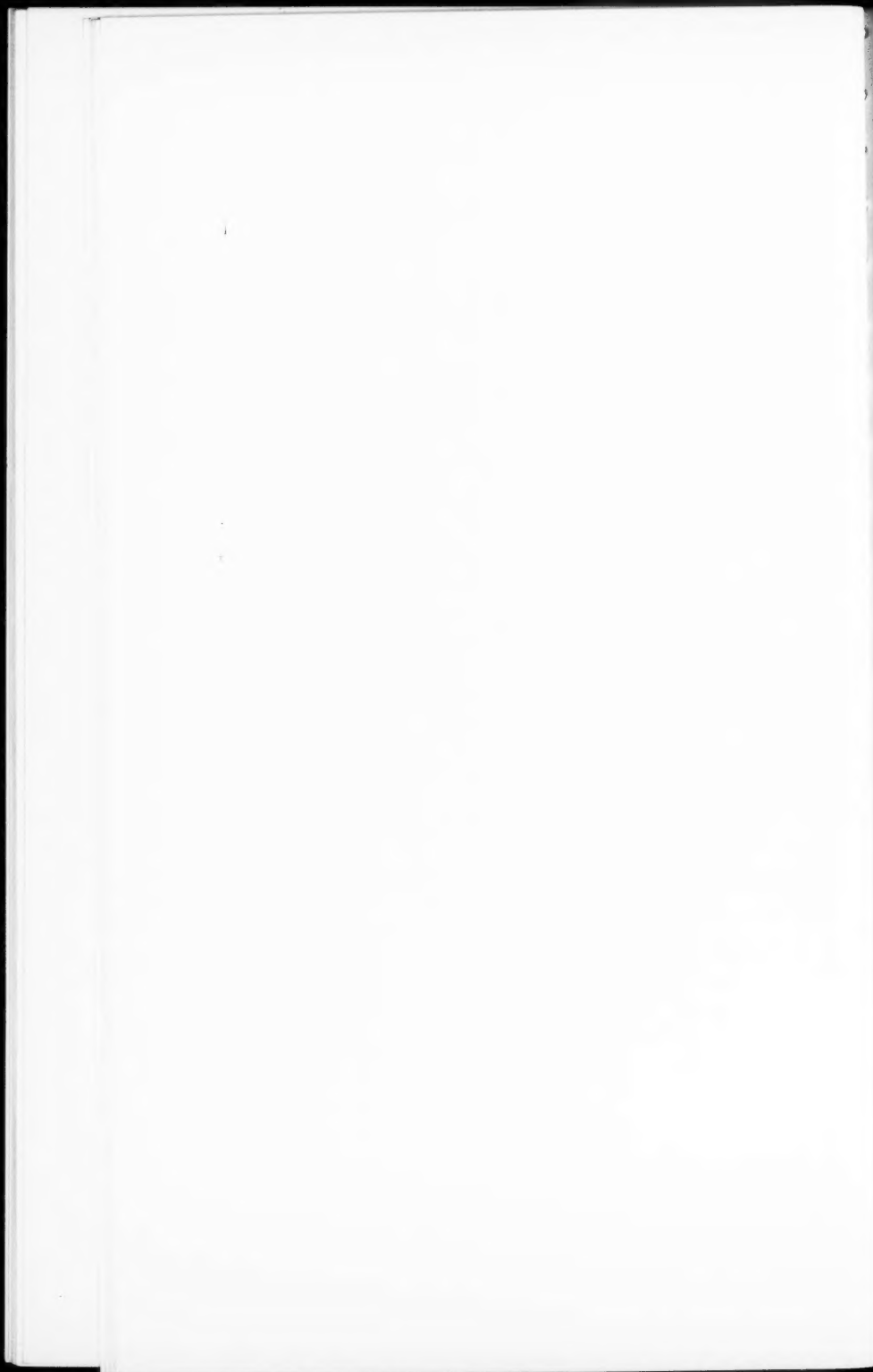
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Synopsis of Technique for Functional Tests

Test I	Tests III, IV	Test V	Test VIII
Pulse Rate	<i>Seconds</i>		<i>Seconds</i>
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60
Diastolic Pressure	Deflate Record	Record Remove Cuff	70-90
(4th Point)	55-60 Inflate "Inspire!"		Standard Exercise
	60 "Blow!"		60 "Blow!"
	60-20 Systolic drop to Minimum	Tests VI, VII	60-60 Heart Rate (per 5 sec.)
	Deflate Record	30-55 Heart Rate (per 5 sec.)	60-60 Heart Rate (per 5 sec.)
Test II	25-15 Inflate—T ₂₀ in seconds	Record every 5 seconds	Record every 5 seconds
	Maximum Systolic	60 "Blow!"	Underline End of Blow
Vital Capacity	Total Length of Blow	60-60 Heart Rate (per 5 sec.)	
Seconds × 0.2 = Liters		Record every 5 seconds	
<i>Repeat</i>	<i>Repeat</i>	Underline End of Blow	Continue counting till original rate is reached

RECORD OF TESTS

Name M. R. C. B Age 53 Sex F Occupation Housewife Ht. 5 ft. 3 ins. Wt. 120 lbs.

I		III	IV	V		VI	VII	VIII
At Rest	Systolic Response- Flamimeter			At Rest	Heart Rate Response - Flamimeter			Number Ascents
					Seconds Before	During and After Blow	During and After Blow	During and After Blow
Pulse Rate	96				0-5	6	8	
					-10	7	8	7
Systolic	158	Systolic	164	154	-15	8	8	7
		Minimum Systolic	160	146	-20	7	7	8
Diastolic	96	Diastolic			-25	7	8	8
		Ten Seconds	—	33	-30	7	8	—
Vital Capacity		Maximum Systolic	174	198	-35	8	7	7
Sec.		Total Seconds	20	40	-40	7	5	8
1					-45	6	8	8
2					-50	6	8	7
					-55	6	8	7
					-60	7	7	7
These lines for Home Office use only								
% of Normal Vital Capacity	0	S+—	4	16	Length of Blow	20	Sec.	40 Sec.
		S—	10	33				Sec.

Remarks:

Was afraid of test and could not blow through 200 cc. orifice sufficiently to raise water to level of glass.

Frequent extra systoles and intermissions.

Very nervous. Note improvement in length of blow in IV as she became reassured.

Auscultation gives no evidence of heart disease. No hypertrophy.

E.K.- Record badly distorted by induction left axial rotation, left ventricular extra systole.

Flarimeter- V.C. not obtained. S- Marked, though in first blow pathological.

S+Normal. Both blows considered S+subnormal. T80 Absent in first blow, subnormal in second. Tm. On borderline between subnormal and pathological. Test revealed distinct lack of myocardial tone.

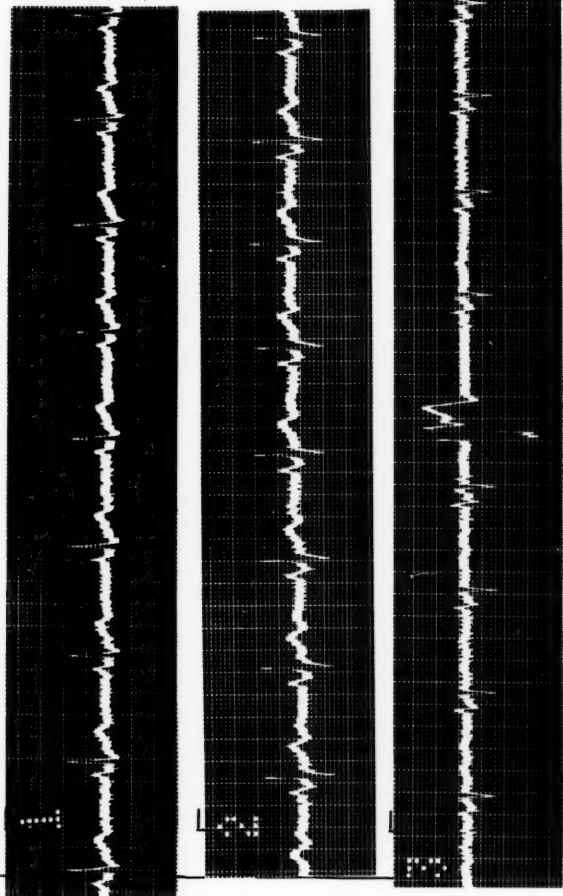
Examiner _____ Residence _____ Date 8/5/29 Hour 2.45 P.M.

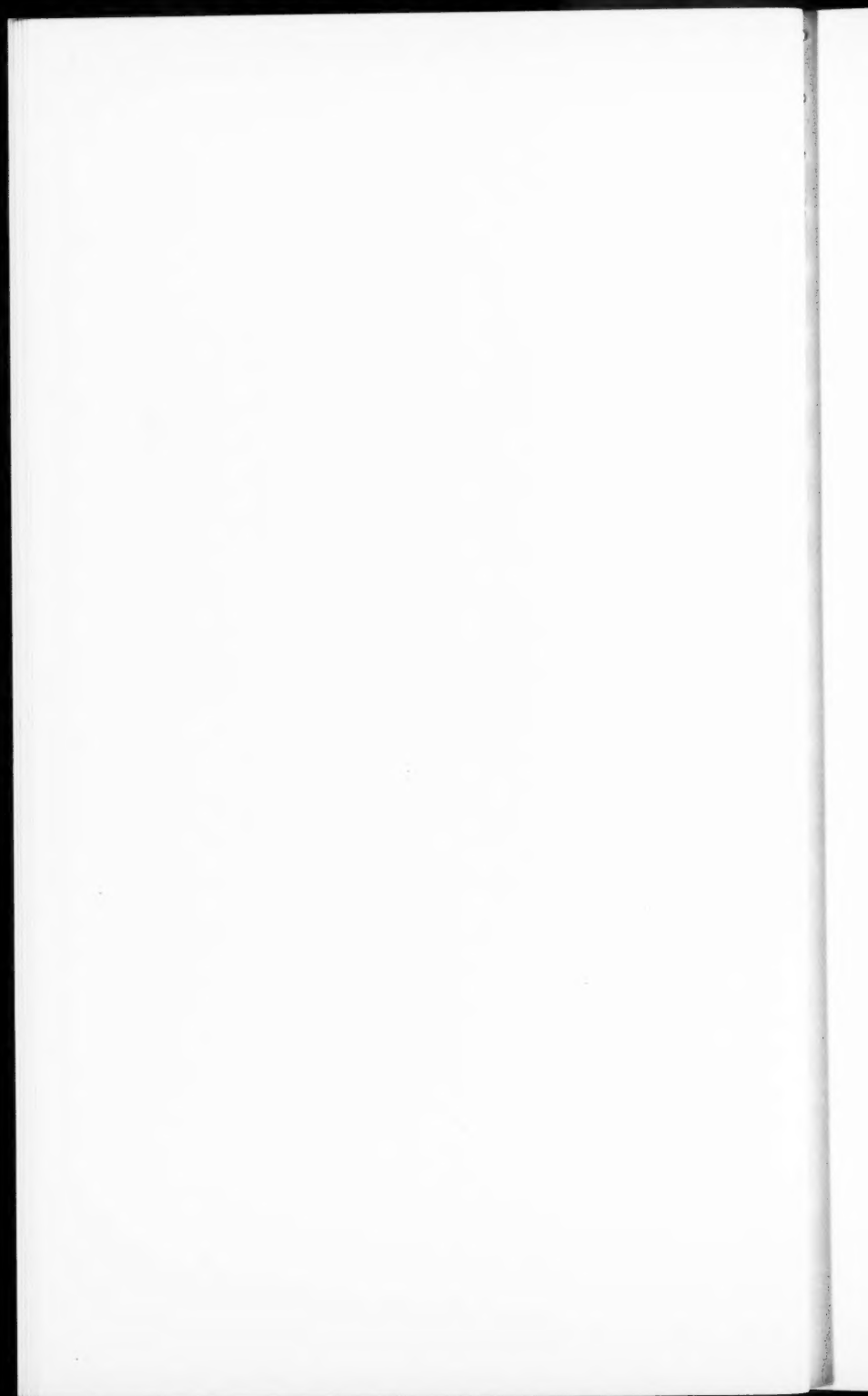
100 M.B. 5-21-29 830 PM

100 M.B. 5-21-29 830 PM

Taken by

Interpreted by





Synopsis of Technique for Functional Tests

Test I

Pulse Rate
Systolic Pressure
Diastolic Pressure
(4th Point)

Tests III, IV

Seconds
25-45 **Systolic Pressure**
Deflate Record
55-60 Inflate "**Inspire!**"
60 "**Blow!**"
60-20 Systolic Drop to **Minimum**
Deflate Record
25-15 Inflate—**T20** in seconds
Maximum Systolic
Total Length of Blow

Test V

Seconds
30-60 **Systolic and Diastolic**
Record Remove Cuff

Tests VI, VII
30-55 **Heart Rate** (per 5 sec.)
Record every 5 seconds
60 "Blow!"
60-60 **Heart Rate** (per 5 sec.)
Record every 5 seconds
Underline End of Blow
Repeat

Test VIII

Seconds
20-60 } **Standard Exercise**
60-50 }
60 "Blow!"
60-60 } **Heart Rate (per 5 sec.)**
60-60 }
Record every 5 seconds
Underline End of Blow

Continue counting till original
rate is reached

Test II

Vital Capacity
Seconds $\times 0.2 =$ Liters
Repeat

Repeat

Repeat

RECORD OF TESTS

Name D. F. C. O Age 39 Sex F Occupation Housewife Ht. 5 ft. 3 ins. Wt. 110 lbs.

I		III	IV	V		VI		VII		VIII	
At Rest		Systolic Response- Phlarmometer		At Rest		Heart Rate Response-Phlarmometer				Number Ascents	
						Seconds	Before	During and After Blow	During and After Blow	During and After Blow	
Pulse Rate	124					0-5			8	7	
						-10			10	8	
						-15			10	10	
Systolic	160	Systolic		Systolic		-20	10	15	10	10	
		Minimum		Diastolic		-25	11	11	10	10	
Diastolic	124	Systolic				-30	10	12	11	10	
		Ta				-35	10	11	9	8	
		Seconds				-40	10	10	9	9	
II		Maximum				-45	10	10	10	9	
Vital Capacity		Systolic				-50	11	10	10	10	
Sec.		Total				-55	-	10	-	8	
1	12	24				-60					
		Libers				-65					
		Seconds				-70					
2	12	24				-75					
						-80					
						-85					
						-90					
						-95					
						-100					
						-105					
						-110					
						-115					
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						-1025					
						-1030					
						-1035					
						-1040					
						-1045					
						-1050			</		

Remarks

Apex 9 cm. from midsternal line. Pregnancy suspected. Rheumatic Mitral stenosis and insufficiency. Systolic thrill at apex.

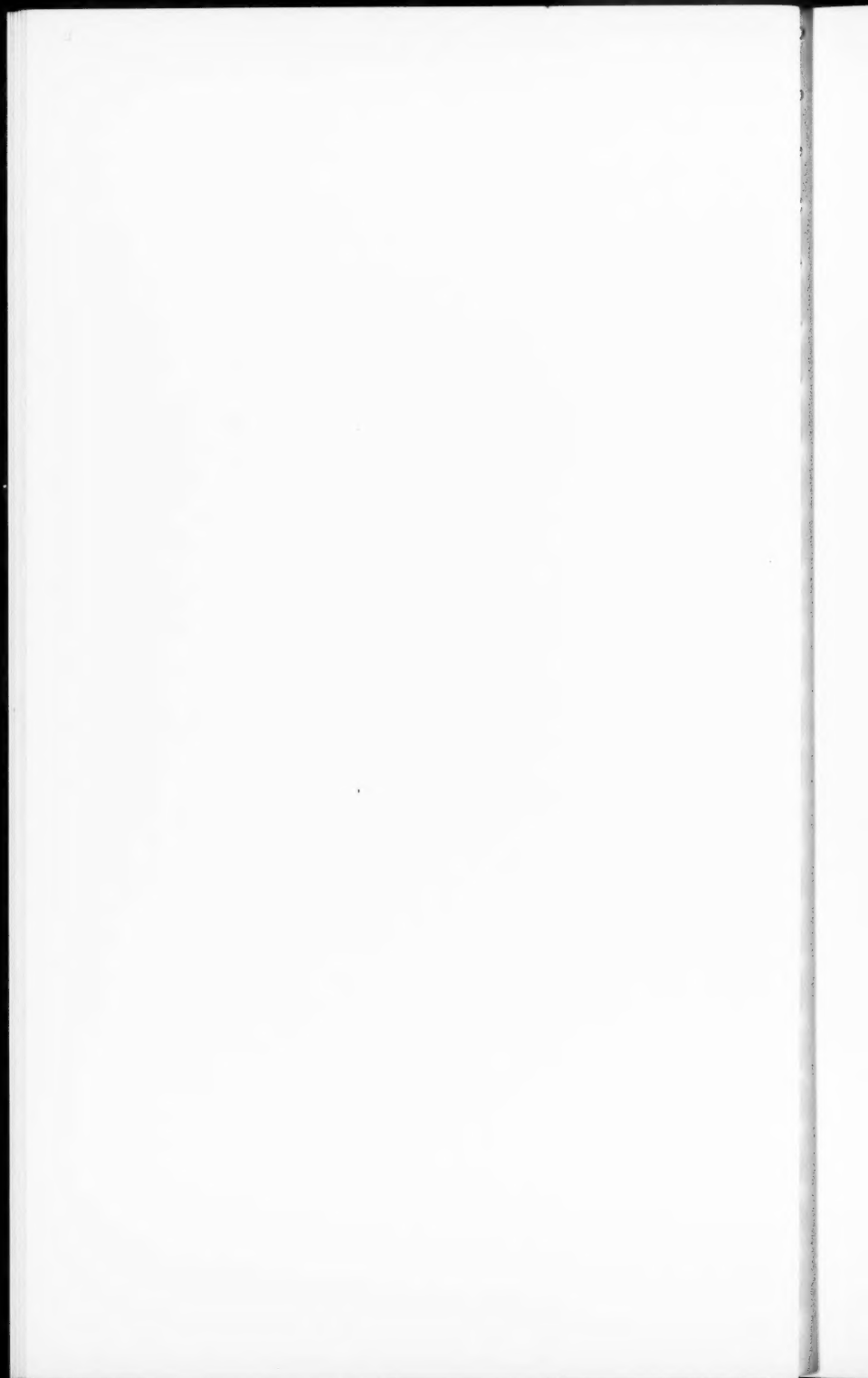
E.K.—Normal sinus rhythm tachycardia.

Flarimeter- V.C. Markedly pathological. S- Marked. S+ Pronouncedly subnormal.

T20 Pronouncedly subnormal. In Pathological

Test shows marked impairment of heart.

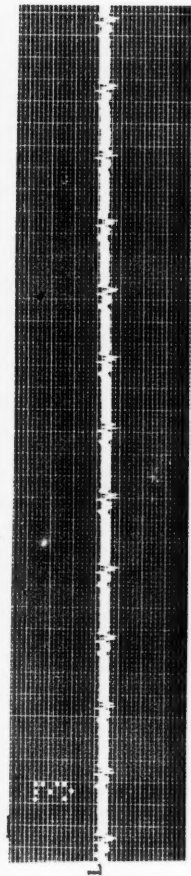
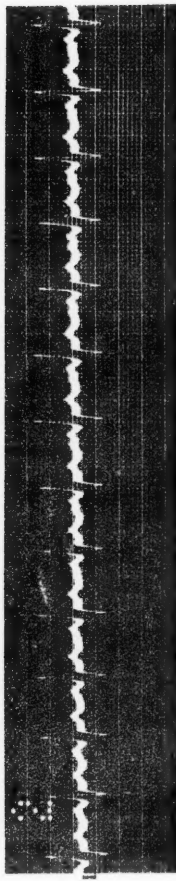
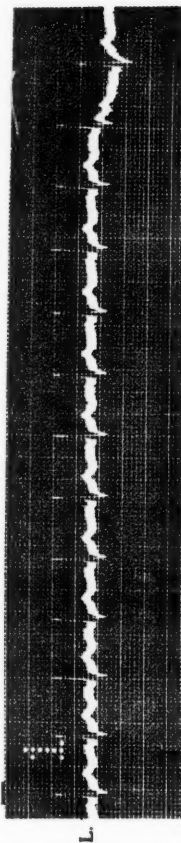
Examiner _____ Residence _____ Date 8/12/29 Hour 9.15 p.m.

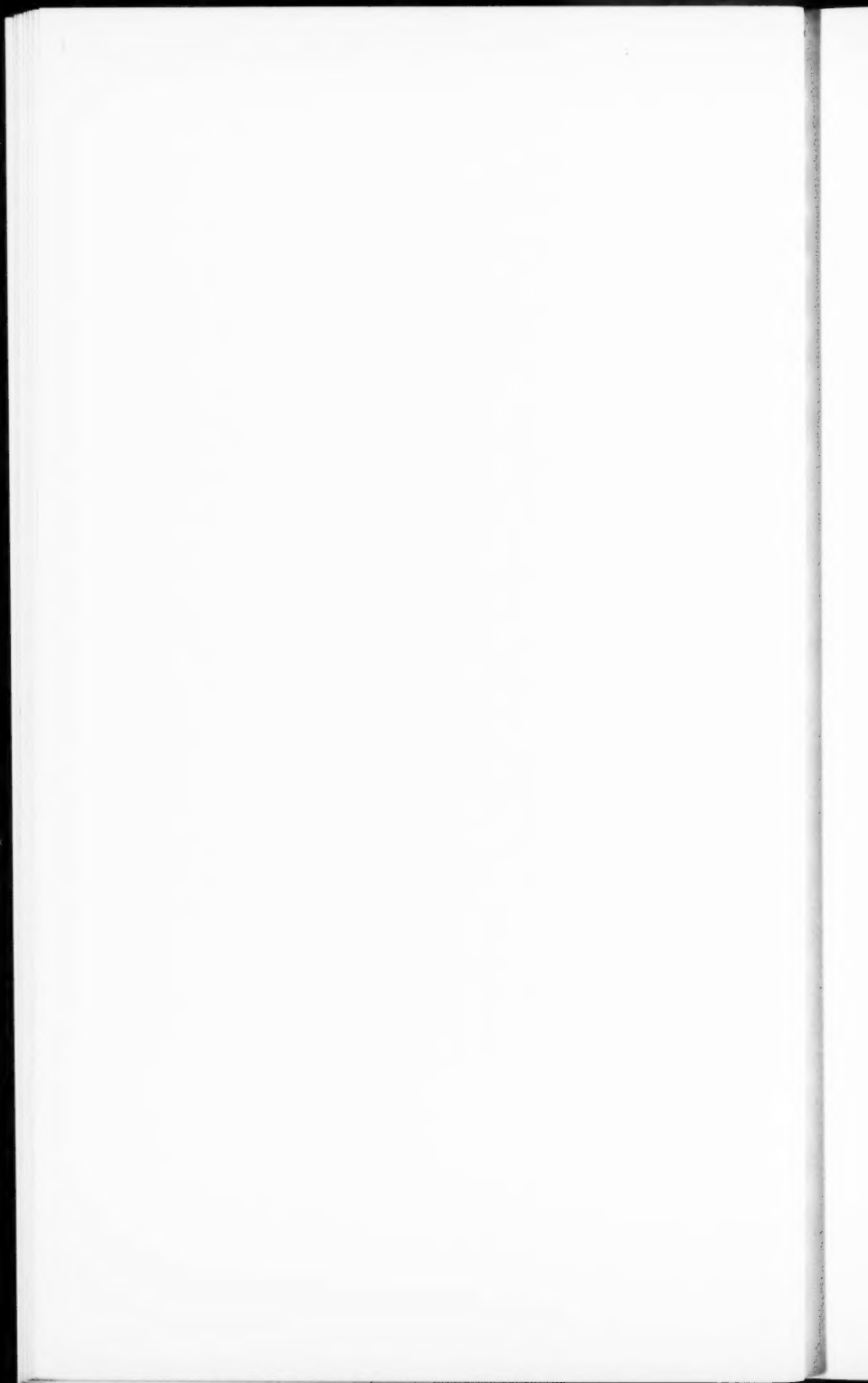


45 D.F. 8-15-29 1 PM

Taken by

Interpreted by





Synopsis of Technique for Functional Tests

Test I	Tests III, IV	Test V	Test VIII
Pulse Rate	Seconds	Seconds	Seconds
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60 Standard Exercise
Diastolic Pressure	Deflate Record	Record Remove Cuff	60-50
(4th Point)	55-60 Inflate "Inspire!"		60 "Blow!"
	60 "Blow!"	Tests VI, VII	60-60 Heart Rate (per 5 sec.)
	60-20 Systolic Drop to Minimum	30-55 Heart Rate (per 5 sec.)	60-60 Heart Rate (per 5 sec.)
	Deflate Record	Record every 5 seconds	Record every 5 seconds
Test II	25-15 Inflate—T20 in seconds	60 "Blow!"	Underline End of Blow
Vital Capacity	Maximum Systolic	60-60 Heart Rate (per 5 sec.)	
Seconds X 0.2 = Liters	Total Length of Blow	Record every 5 seconds	
Repeat	Repeat	Underline End of Blow	Continue counting till original rate is reached
		Repeat	

RECORD OF TESTS

Name A. J. C. R. Colored Age 36 Sex M Occupation Laborer Ht. 5 ft. 9 ins. Wt. 130 lbs.

I		III		IV	V	VI				VII		VIII
At Rest		Systolic Response-Flameter			At Rest	Heart Rate Response - Flameter				After		Number
						Seconds	Before	During and After Blow	During and After Blow			Amounts
Pulse Rate	65					0-5		5	4	6	5	
Systolic	146	Systolic	146	140	Systolic	-10		5	5	6	4	
Diastolic	118	Minimum Systolic	130	128	Diastolic	-15		5	5	7	5	
		Seconds	35	35		-20		7	5	7	5	
		Maximum Systolic	178	166		-25		7	5	8	5	
Vital Capacity		Seconds	35	35		-30		8	5	8	5	
Sec.		Total	48	40		-35		8	5	7	5	
1	15	3.0				-40	5	8	4	8		
2	17	3.4				-45	5	8	5	6		
						-50	6	8	5	5		
						-55	5	5	6	4		
						-60	5	4	-	4		
These lines for Home Office use only						Length of Blow		40	Sec.	40	Sec.	Sec.
% of Normal Vital Capacity	78	S-	16	14								
		S+	26	26								

Remarks:

Maximum apical impulse 10cm. from midsternal line.

Clinical diagnosis- mitral stenosis - rheumatic.

E. E. Normal. Dr. Folevski.

Former long distance runner up to 20 miles.

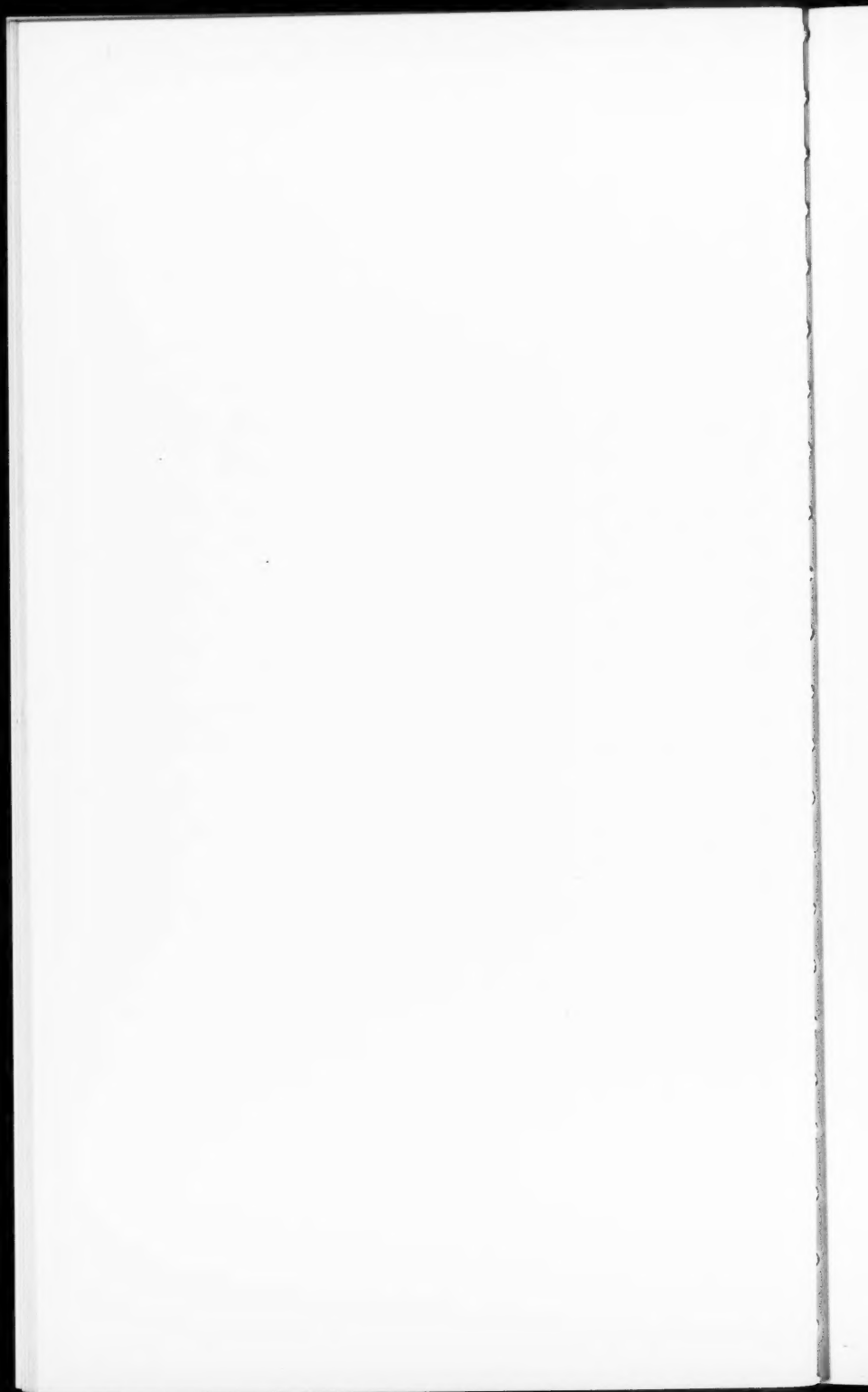
Present complaints largely psychic.

When seen one week later claimed test had helped him.

Flameter- V.C. Pathological. S- Marked. S+ Subnormal. T20 Normal. Tm Normal.

This man was afraid to blow owing to precordial pain. Several other readings gave the diastolic as 100. The man really tried to do his best. The test points to a somewhat more favorable condition than does his vital capacity and high diastolic pressure, in spite of the presence of mitral stenosis, with a slight hypertrophy.

Examiner.....Residence.....Date 7/30/29 Hour 9 P.M.



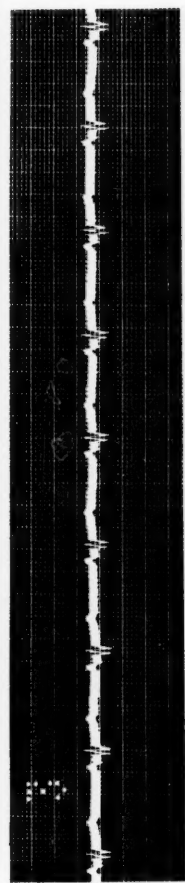
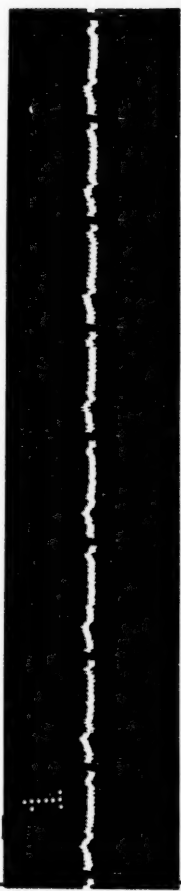
125 A.J. 1-23-29 8 P.M.

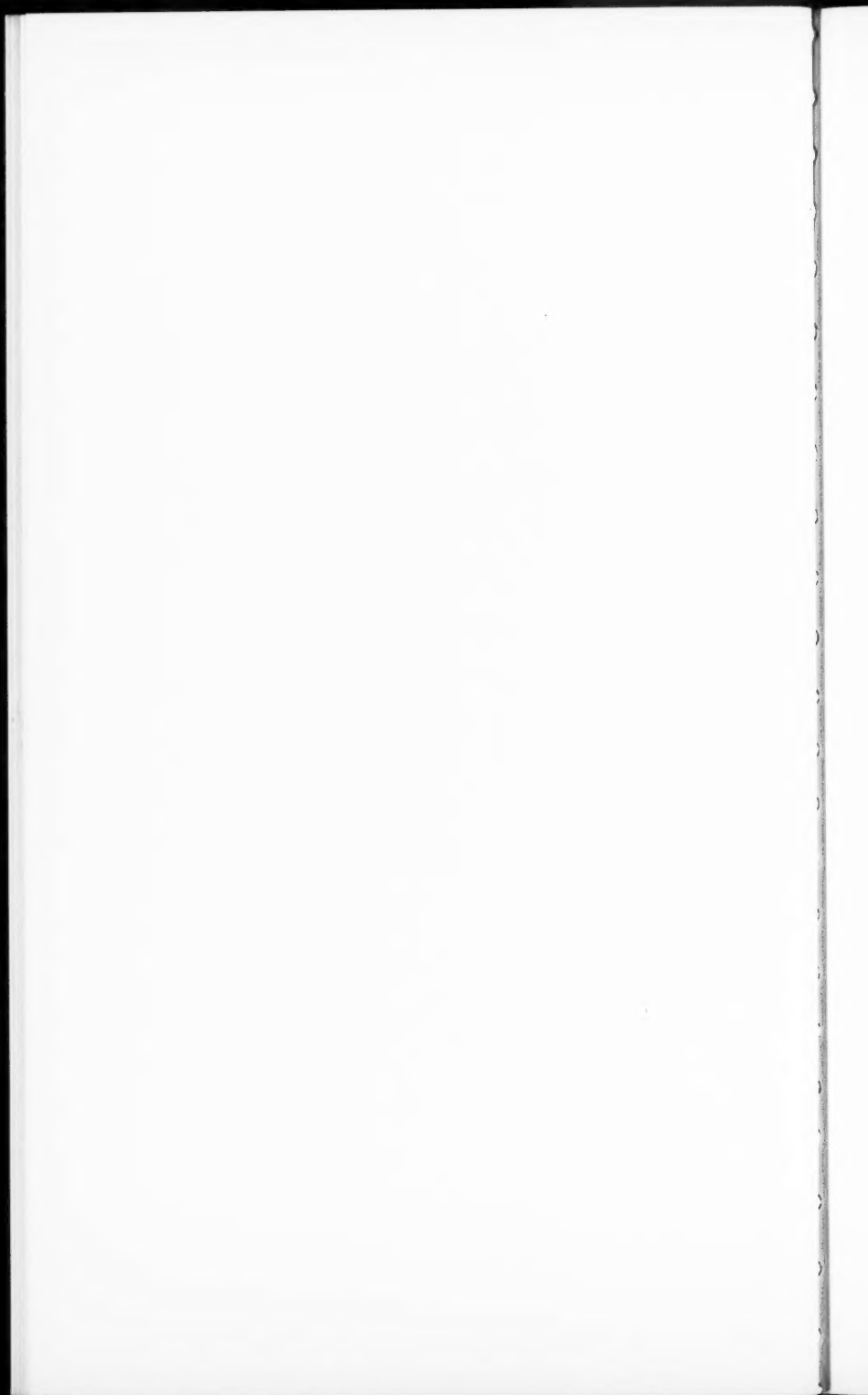
Taken by

7 Tracy

Interpreted by

W. H. H. H.





Synopsis of Technique for Functional Tests

Test I	Tests III, IV	Test V	Test VIII
Pulse Rate	Seconds	Seconds	Seconds
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60 Standard Exercise
Diastolic Pressure	Deflate Record	Record Remove Cuff	60-50 "Blow!"
(4th Point)	55-60 Inflate "Inspire!"		60 "Blow!"
	60 "Blow!"	Tests VI, VII	60-60 Heart Rate (per 5 sec.)
	60-20 Systolic Drop to Minimum	30-55 Heart Rate (per 5 sec.)	Record every 5 seconds
	Deflate Record	Record every 5 seconds	Underline End of Blow
Test II	25-15 Inflate—T20 in seconds	60 "Blow!"	
Vital Capacity	Maximum Systolic	60-60 Heart Rate (per 5 sec.)	
Seconds X 0.2 = Liters	Total Length of Blow	Record every 5 seconds	
Repeat	Repeat	Underline End of Blow	Continue counting till original rate is reached

RECORD OF TESTS

Name **A. M.** Age **21** Sex **F** Occupation **...** Ht. **5** Ft. **3** Ins. **120** Wt. **120** lbs.

I		III		IV	V	VI		VII	VIII
At Rest		Systolic Response-Fluorimeter			At Rest	Heart Rate Response-Fluorimeter		After	Number Ascents
						Seconds Before	During and After Blow	During and After Blow	
Pulse Rate	100					0-5	8 8	8 8	
Systolic	138	Systolic	130	130	Systolic	10	11 8	9 8	
Diastolic	72	Minimum Systolic	110	108	Diastolic	15	11 9	12 9	
		T20 Seconds	-	-		20	10 8	10 8	
		Maximum Systolic	164	146		25	11 8	11 8	
Vital Capacity		Total Seconds	25	27		30	7 7	8 10	8
Sec.						35	7 7	8 8	
1	12	E. 4				40	10 8	10 8	
2	11	E. 2				45	9 9	9 8	
These lines for Home Office use only									
% of Normal Vital Capacity	70	S-	20	24		50	- 8	8 8	
		S+	28	18		55	- 9	- 9	
						60	22	27	
						Length of Blow	Sec.	Sec.	Sec.

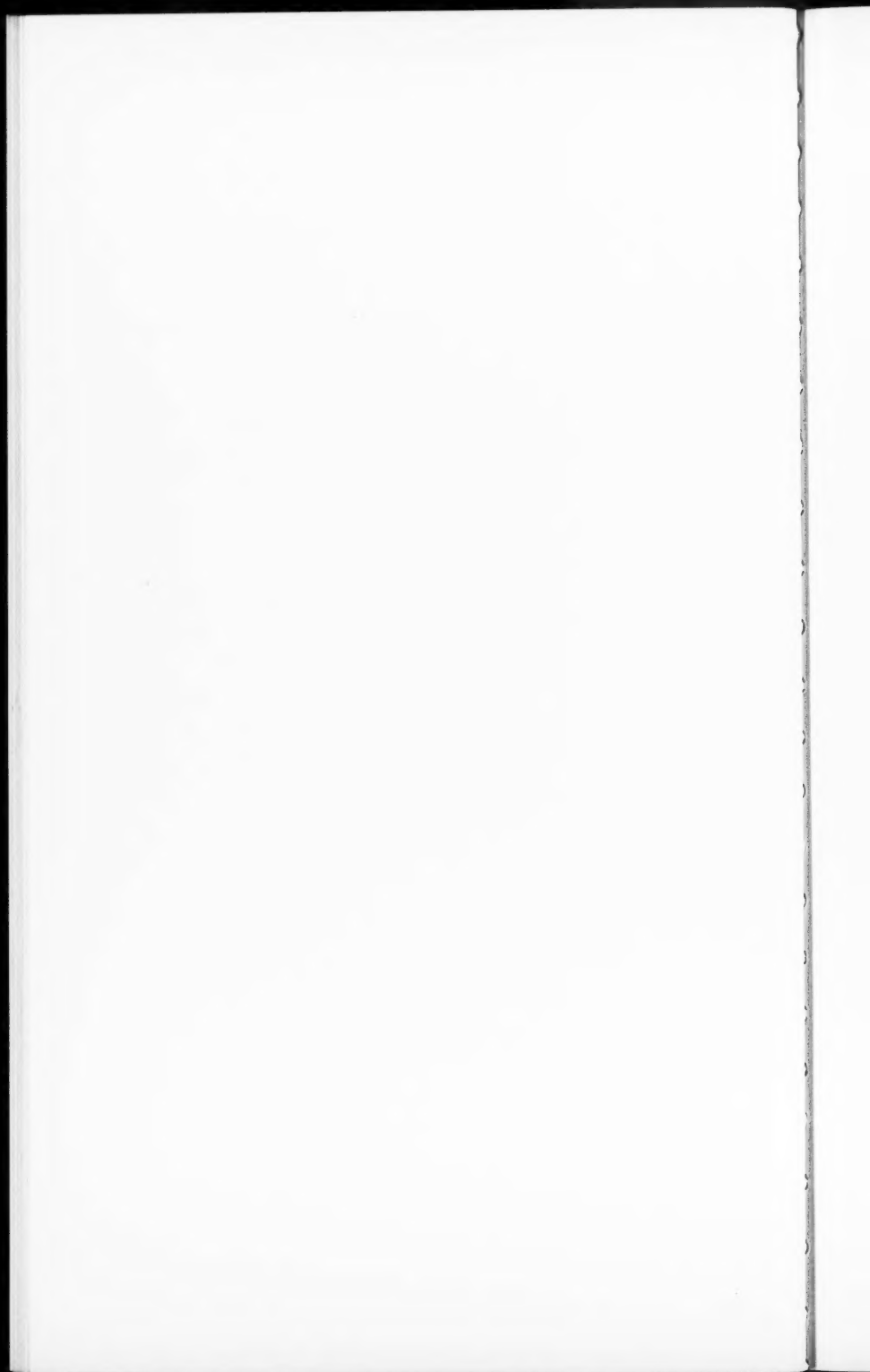
Remarks: Apex impulse 10 cm. from midsternal line. Slight hypertrophy. Double mitral murmur - rheumatic.

E.K.- Seems normal but for tachycardia.

Fluorimeter- V. C. Pathological. S- Marked. S+ Normal.
T20 Absent T25 Pathological

Test indicates decided impairment of heart.

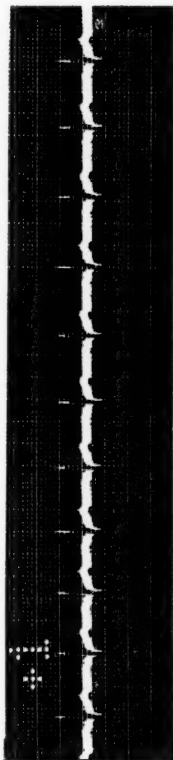
Examiner **...** Residence **...** Date **8.19.29** Hour **9.30** P.M.
9800



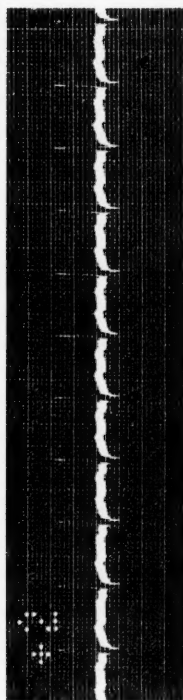
76 AM. 4-16-29 830 PM.

Taken by _____

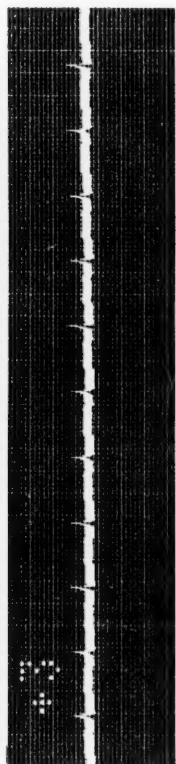
Interpreted by _____



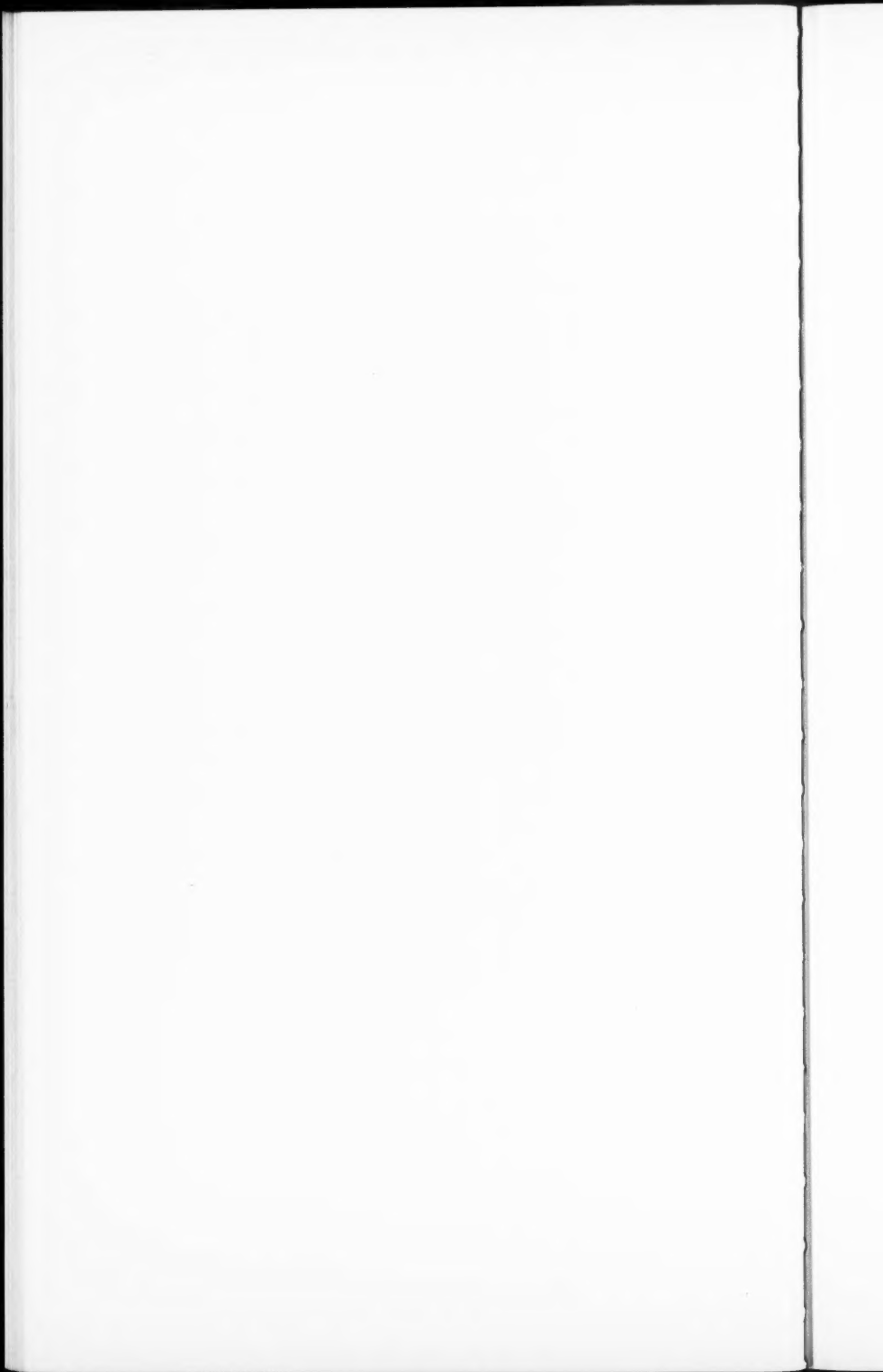
L.1



L.2



L.3



Synopsis of Technique for Functional Tests

Test I Pulse Rate Systolic Pressure Diastolic Pressure (4th Point) Test II Vital Capacity Seconds $\times 0.2 =$ Liters Repeat	Tests III, IV Seconds 25-45 Systolic Pressure Deflate Record 55-60 Inflate "Inspire!" 60 "Blow!" 60-20 Systolic Drop to Minimum Deflate Record 25-15 Inflate—T20 in seconds Maximum Systolic Total Length of Blow Repeat	Test V Seconds 30-60 Systolic and Diastolic Record Remove Cuff Tests VI, VII 30-55 Heart Rate (per 5 sec.) Record every 5 seconds 60 "Blow!" 60-60 Heart Rate (per 5 sec.) Record every 5 seconds Underline End of Blow Repeat	Test VIII Seconds 20-60 Standard Exercise 60-30 "Blow!" 60-60 Heart Rate (per 5 sec.) Record every 5 seconds Underline End of Blow Continue counting till original rate is reached
--	--	---	---

RECORD OF TESTS

Name: E. P. A. C. 6 Age: 42 Sex: 7 Occupation: Housework Ht. 5 ft. 2 ins. Wt. 155 lbs.

I		III		IV	V	VI		VII	VIII	
At Rest		Systolic Response- Fluorimeter			At Rest	Heart Rate Response- Fluorimeter			Number	Ascents
						Seconds	Before	During and After Blow	During and After Blow	During and After Blow
Pulse Rate	56					9-3		7	4	
Systolic	156	Systolic	166	166	Systolic	-10	4	-	7	
Diastolic	82	Minimum Systolic	-	146	Diastolic	-15	4	5	6	
II		T20	-	-		-20	6	4	6	
Vital Capacity		Seconds	-	-		-25	6	5	4	
Sec.		Maximum Systolic	178	178		-30	7	5	6	
1	11 8.2	Total Seconds	36	36		-35	7	-	7	
2	13 8.6					-40	7	5	7	
These lines for Home Office use only										
% of Normal Vital Capacity	90	S-	-	20		-45	5	4	7	
		S+	12	6		-50	6	5	7	
						-55	3	7	-	6
						-60	3	7	-	5
						Length of Blow	36	Sec.	36	Sec.

Remarks:

Pulse so irregular that rate and blood pressure were very difficult to obtain.
Taking digitalis. History of acute inflammatory rheumatism and tonsillitis.
Diagnosis

E.K.- Auricular fibrillation advanced. Coarse circus movement, left axial rotation.

Fluorimeter- V.C. Normal. S- Missed, though marked in second blow. S+ markedly pathological. T20 Absent. Tm Pathological.

The second test was even worse than the first, and both pointed to a very serious condition.

Examiner: _____ Residence: _____ Date: 9/4/29 Hour: 8:30 P.M.
79028



95 E.P. 5-14-29 830 P.M.

same as before

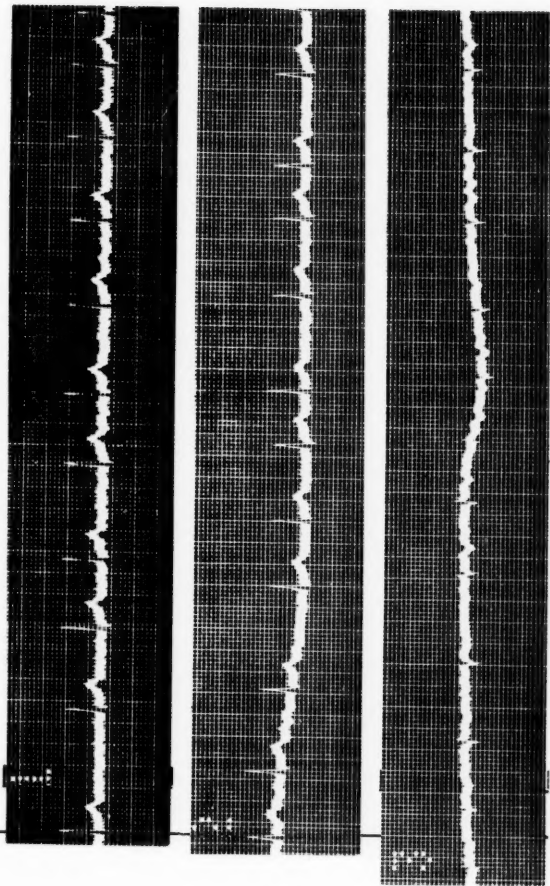
light as in before

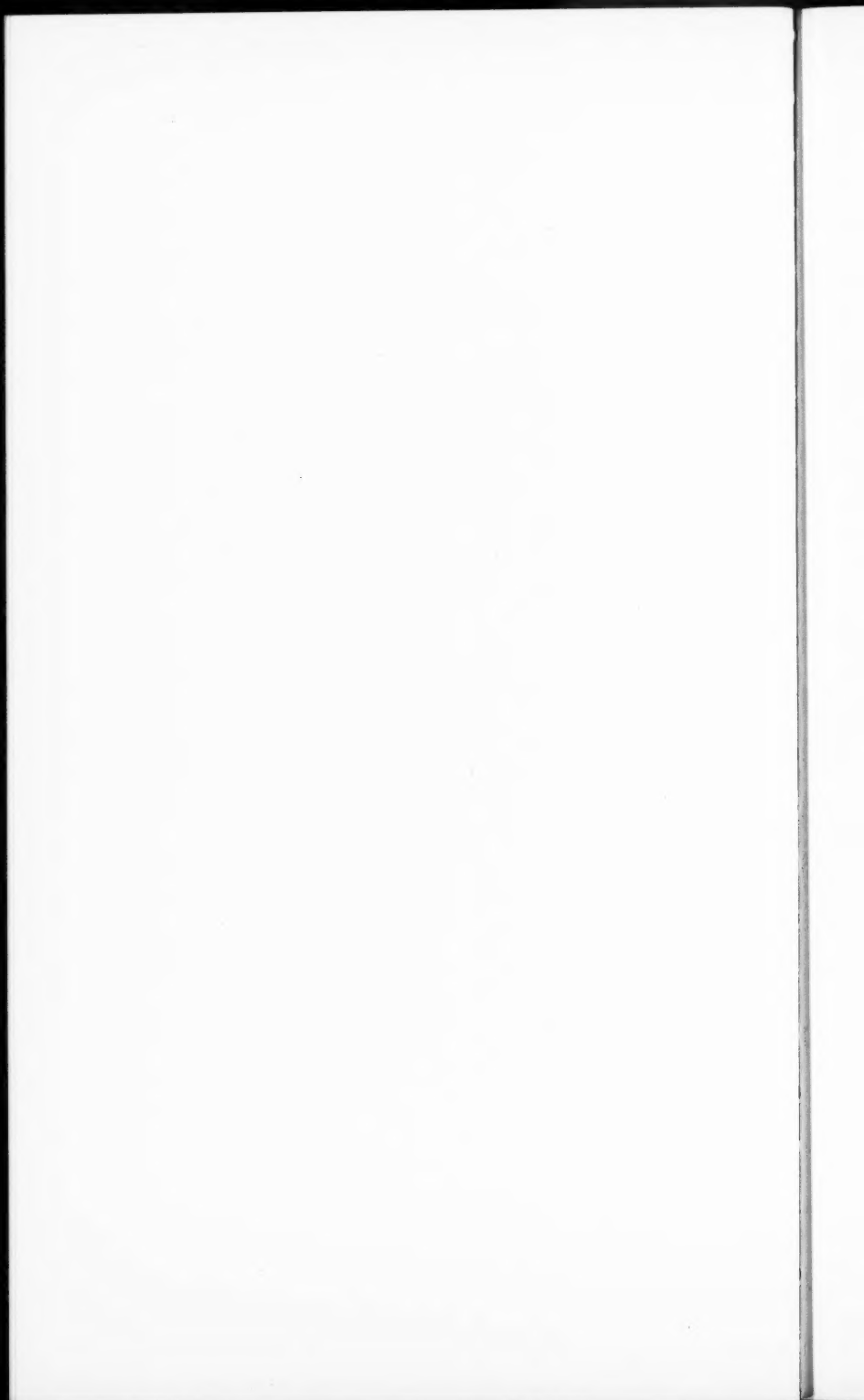
Taken by

7 May

Interpreted by

Max count





Synopsis of Technique for Functional Tests

Test I	Tests III, IV	Test V	Test VIII
Pulse Rate	Seconds	Seconds	Seconds
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60 Standard Exercise
Diastolic Pressure	Deflate Record	Record Remove Cuff	60-50 "Blow!"
(4th Point)	55-60 Inflate "Inspire!"	Tests VI, VII	60 "Blow!"
	60 "Blow!"	30-55 Heart Rate (per 5 sec.)	60-60 Heart Rate (per 5 sec.)
	60-20 Systolic Drop to Minimum	Record every 5 seconds	Record every 5 seconds
	Deflate Record	60 "Blow!"	Underline End of Blow
Test II	25-15 Inflate-T20 in seconds	60-60 Heart Rate (per 5 sec.)	
Vital Capacity	Maximum Systolic	Record every 5 seconds	
Seconds $\times 0.2 =$ Liters	Total Length of Blow	Underline End of Blow	
Repeat	Repeat	Repeat	Continue counting till original rate is reached

RECORD OF TESTS

Name M. S. J. G. 3702 Age 16 Sex M Occupation Ht 5 ft 2 3/4 in. Wt 120 lbs.

I		III IV		V	VI				VII	VIII	
At Rest		Systolic Response-Fluimeter		At Rest	Heart Rate Response - Fluimeter					Number Ascents	
					Seconds	Before	During and After Blow	During and After Blow		During and After Blow	
Pulse Rate	92				0-5		8	7	9		
Systolic	149	Systolic	160 48	Systolic	-10		7	7	9		
Diastolic	120	Minimum Systolic	150 122	Diastolic	-15		9	7	9		
		T20 Seconds	- 35		-20		9	9	9		
		Maximum Systolic	195 172		-25		9	8	10		
		Total Seconds	25 45		-30		10	8	9		
					-35		8	10	8		
					-40		8	9	7		
					-45		7	9	8		
					-50		7	9	7		
					-55		8	9	8		
					-60		-	8	-		
					Length of Blow		45	Sec. 30	Sec.		

These lines for Home Office use only

Remarks: Heart rate tests not simultaneous with blows.

Dyspnea and palpitation.

Chest 28.5 cm. Left heart 10.3. Rt. heart 8.7. Ratio heart to chest 67%.

History rheumatic fever.

Diagnosis- Mitral stenosis and aortic regurgitation.

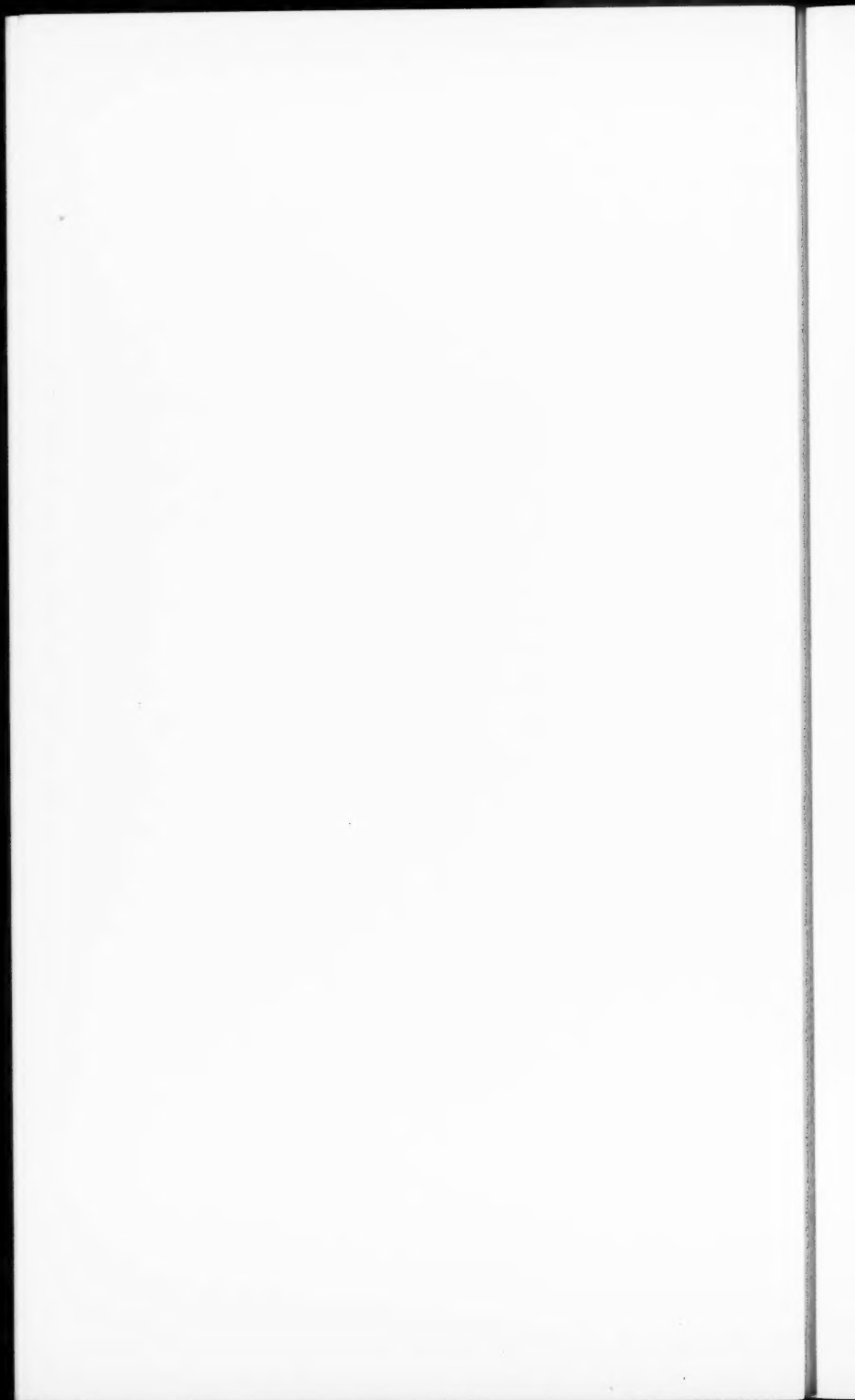
E. K. Toxic tachycardia. Dr. Polevski.

Fluimeter- V. C. Pathological. S- Marked. S+ Subnormal. T20 Subnormal.

T20 Subnormal. Delayed return in heart rate.

The combined tests indicate definite myocardial involvement. The patient was in bed and the resting heart was able to put forth better effort. He cooperated splendidly on the last blow and exerted himself to the maximum of his capacity.

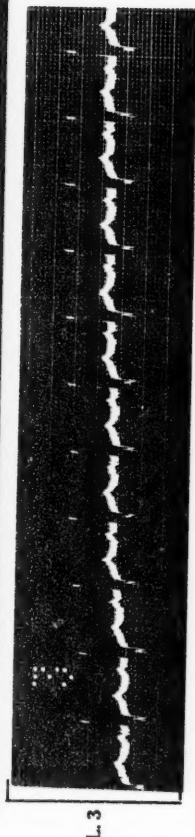
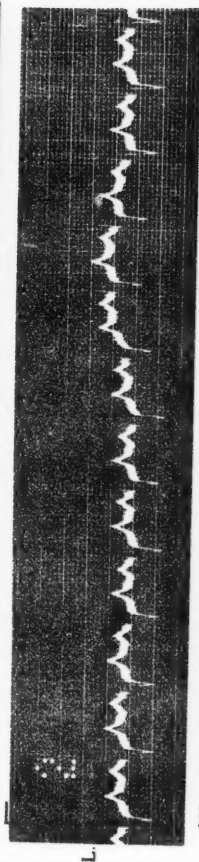
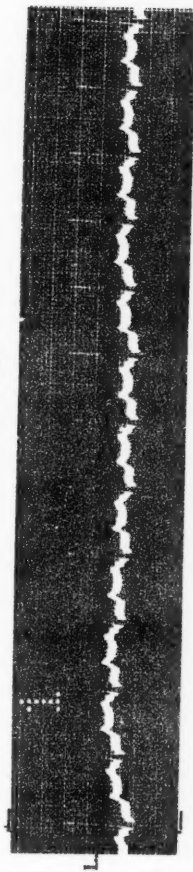
Examiner Residence Date 7/19/29 Hour 10 a.m.
vms

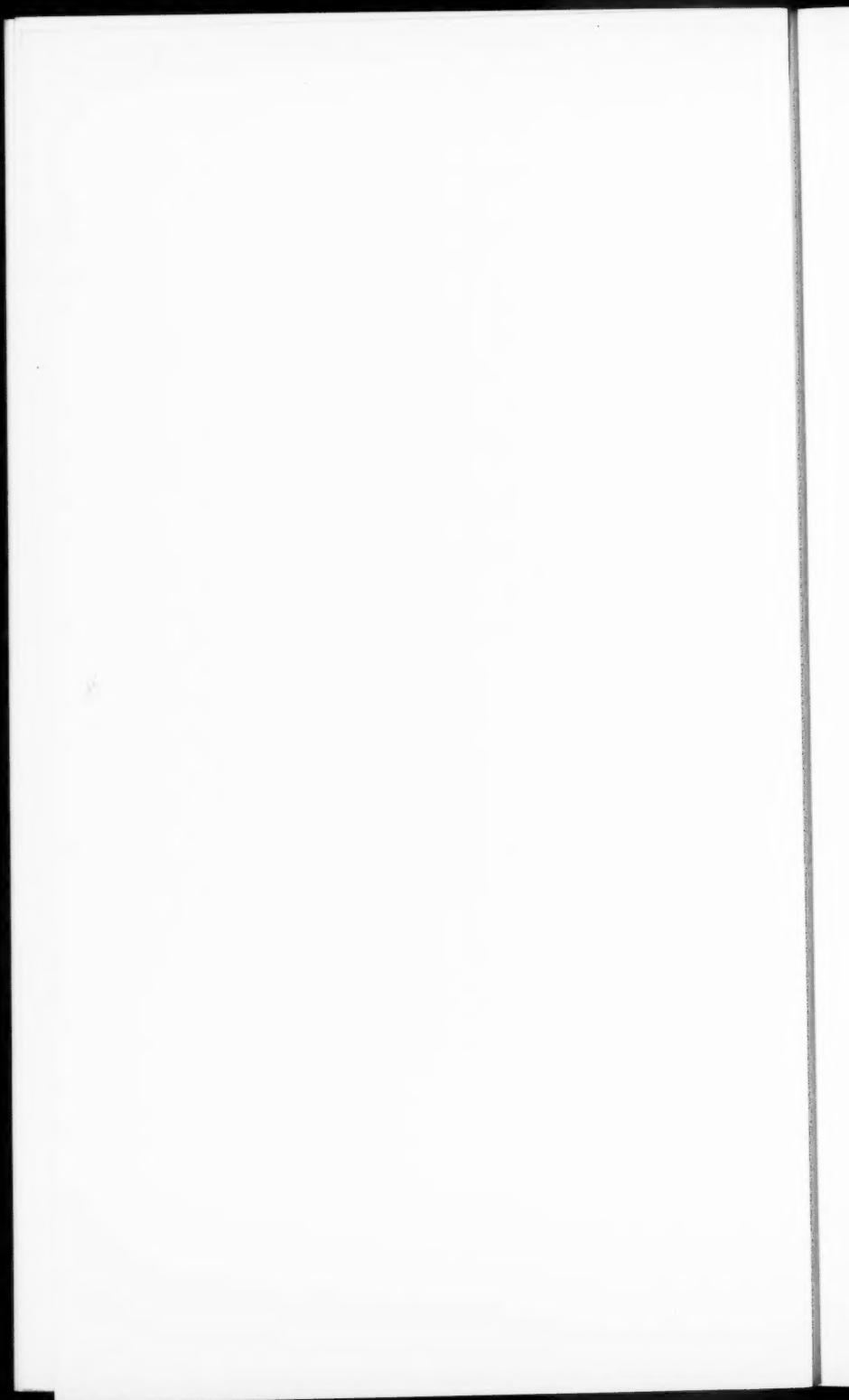


G 3702 M.S. 6-5-29 2 AM

Taken by

Interpreted by





Synopsis of Technique for Functional Tests

Test I	Tests III, IV	Test V	Test VIII
Pulse Rate	Seconds	Seconds	Seconds
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60
Diastolic Pressure	Deflate Record	Record Remove Cuff	60-60) Standard Exercise
(4th Point)	55-60 Inflate "Inspire!"		60-60) 60 "Blow!"
	60 "Blow!"	Tests VI, VII	60-60) Heart Rate (per 5 sec.)
Test II	60-20 Systolic Drop to Minimum	30-35 Heart Rate (per 5 sec.)	60-60) Record every 5 seconds
	Deflate Record	60 "Blow!"	60-60) Underline End of Blow
Vital Capacity	25-15 Inflate—T20 in seconds	60-60) Heart Rate (per 5 sec.)	
Seconds $\times 0.2$ = Liters	Maximum Systolic	60-30) Record every 5 seconds	
Repeat	Total Length of Blow	Underline End of Blow	Continue counting till original rate is reached
	Repeat	Repeat	

RECORD OF TESTS

Name Re. S. a. C. s. Age 15 Sex M. Occupation Ht. 5 ft. 3 in. Wt. 117 lbs.

I		III		IV		V		VI		VII		VIII	
At Rest		Systolic Response-Flamimeter		At Rest		At Rest		Heart Rate Response - Flamimeter		During and After Blow		After Number Ascents	
Pulse Rate	100							Seconds	Before	During and After Blow	During and After Blow	During and After Blow	During and After Blow
Systolic	128	Systolic	158	130	Systolic			0-5		8	8	7	
Diastolic	68	Minimum	134	128	Diastolic			-10		7	8	8	
		Systolic						-15		8	9	8	7
		Minimum						-20		8	8	10	8
		Seconds						-25	8	8	9	9	8
		T20						-30	8	8	10	10	8
Vital Capacity		Maximum	148	158				-35		7	8	9	8
Sec. Liters		Total						-40	9	8	9	8	8
1 13	2.6	Seconds	25	30				-45	8	8	8	8	8
2 12	2.4							-50	9	8	8	7	8
								-55	9	8	7	7	7
								-60	8	8		8	7
								Length of Blow	16	Sec	30	Sec	Sec
These lines for Home Office use only													
% of Normal Vital Capacity	61	S-	5	4									
		S+	0	28									

Remarks: Clinic case. Collapsing pulse. Increase in transverse diameter of heart. Apex downward and to left. Slight bulging in auriculo-pulmonic curve.
History of rheumatism and tonsillitis at age 4. Heart condition discovered at 11 years of age.

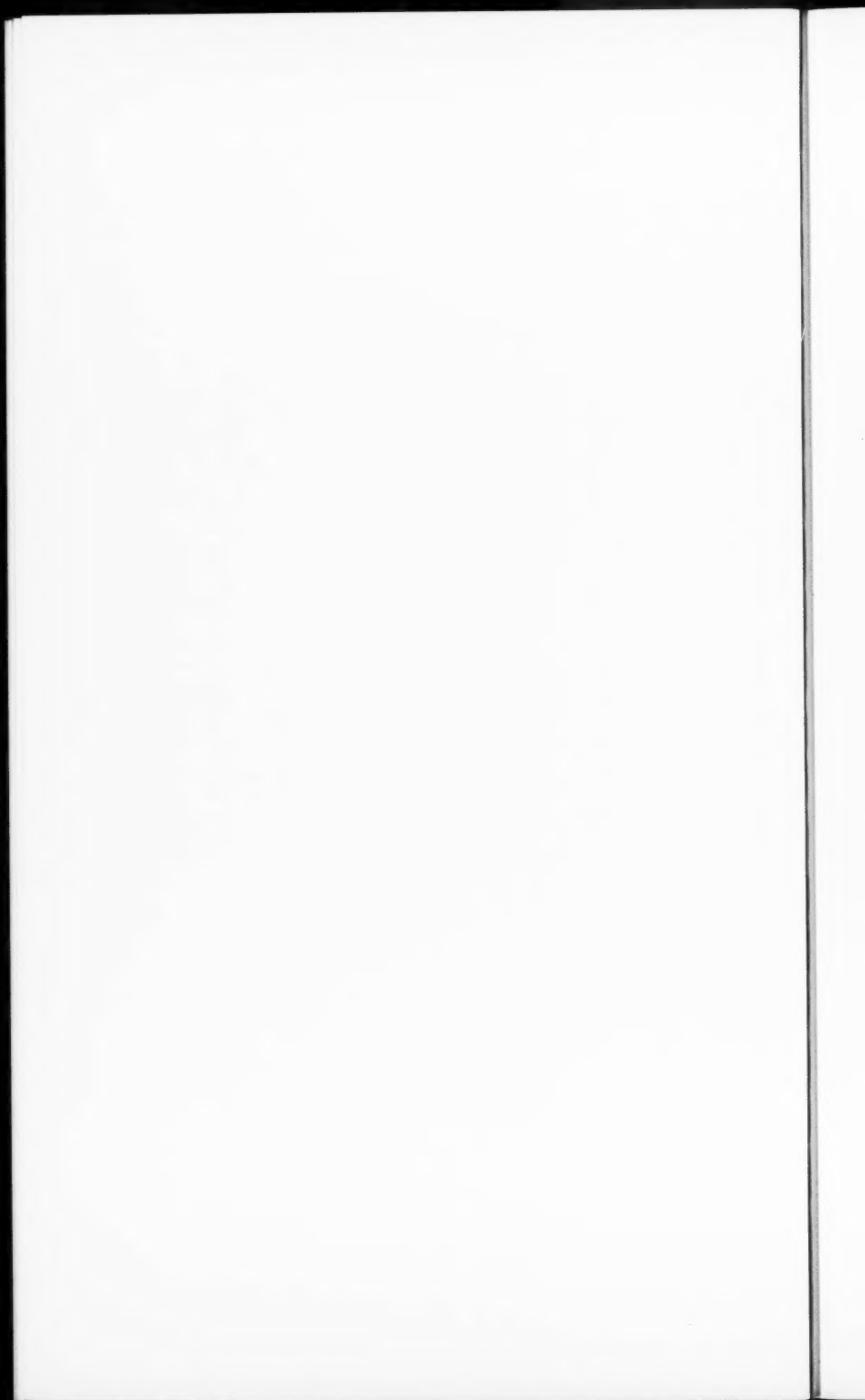
Clinical diagnosis - mitral and aortic regurgitation.

E.K.- Left ventricular preponderance.

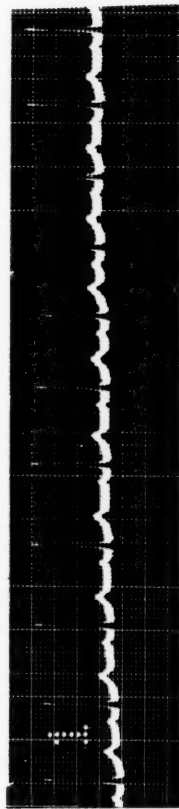
Flamimeter- V. C. Pathological. S- shortened. S-I-Subnormal
T20 Subnormal Tm Pathological

The whole test shows in a very marked way the existence of a pathological condition.

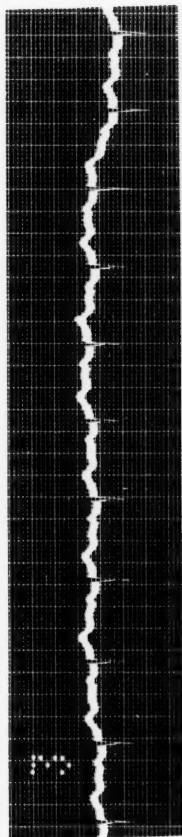
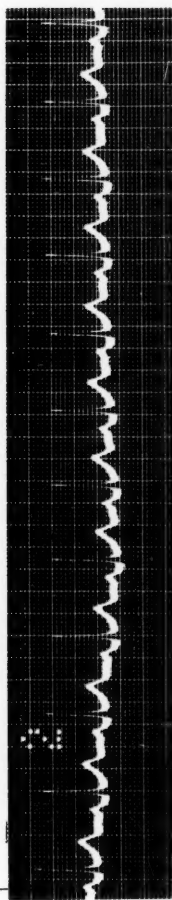
Examiner Residence Date 8/12/29 Hour 3 P.M.



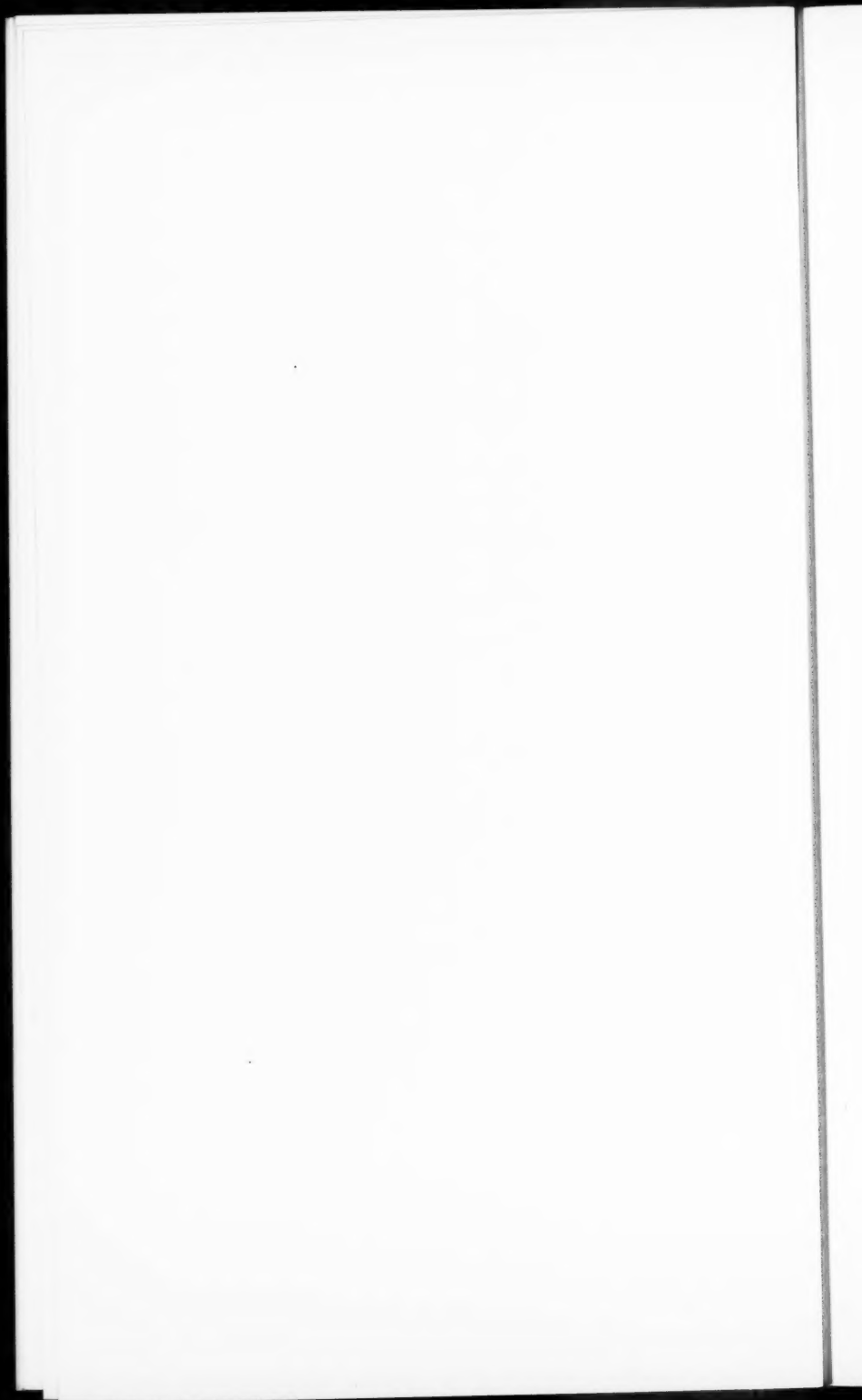
107 R.S. 6-18-29



L1



L1



Synopsis of Technique for Functional Tests

Test I Pulse Rate Systolic Pressure Diastolic Pressure (4th Point)	Tests III, IV Seconds 25-45 Systolic Pressure Deflate Record 55-60 Inflate "Inspire!" 60 "Blow!" 60-20 Systolic Drop to Minimum Deflate Record 25-15 Inflate—T20 in seconds Maximum Systolic Total Length of Blow	Test V Seconds 30-60 Systolic and Diastolic Record Remove Cuff Tests VI, VII 30-55 Heart Rate (per 5 sec.) Record every 5 seconds 60 "Blow!" 60-60 Heart Rate (per 5 sec.) Record every 5 seconds Underline End of Blow Repeat	Test VIII Seconds 20-60 Standard Exercise 60-60 "Blow!" 60-60 Heart Rate (per 5 sec.) Record every 5 seconds Underline End of Blow Continue counting till original rate is reached
Test II Vital Capacity Seconds X 0.2 = Liters Repeat	Repeat	Repeat	Repeat

RECORD OF TESTS

Name S. S. A. C. P. Age 18 Sex F. Occupation Ht. 5 ft. 1 in. Wt. 115 lbs.

I		III		IV		V		VI		VII		VIII	
At Rest		Systolic Response- Fluorimeter		At Rest		Heart Rate Response- Fluorimeter		During and After Blow		During and After Blow		Number After During and After Blow	
Pulse Rate	60					Seconds	Before	During and After Blow	During and After Blow	During and After Blow	During and After Blow	During and After Blow	During and After Blow
Systolic	138	Systolic	152	158	Systolic	0-5	5	5	5	5	5		
Diastolic	52	Minimum Systolic	0	148	Diastolic	-10	5	6	6	4			
		T20 Seconds	18	20		-15	6	5	8	4			
		Maximum Systolic	194	202		-20	8	5	8	5			
		Total Seconds	35	40		-25	8	6	8	5			
						-30	8	5	9	6			
						-35	5	7	5	7	5		
						-40	5	7	5	8	6		
						-45	5	6	6	8	6		
						-50	6	6	6	7	6		
						-55	6	5	6	7	5		
						-60	5	5	-	5	5		
						Length of Blow	35	Sec.	40	Sec.		Sec.	

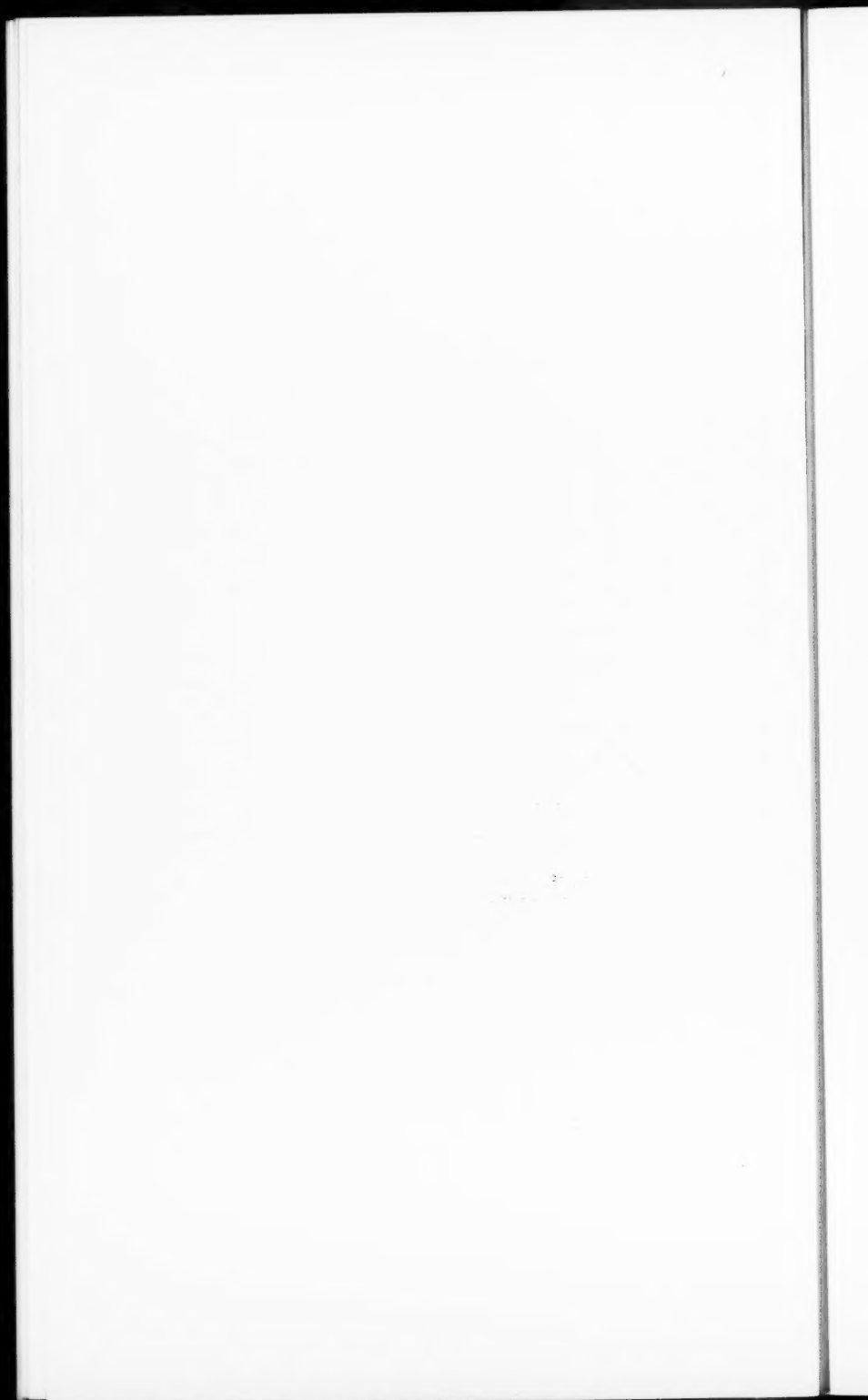
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% of Normal/Vital Capacity 65 5- 0 4

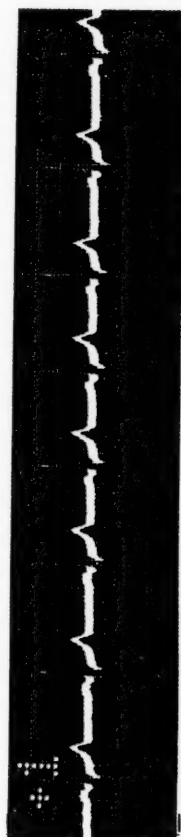
Remarks: Chest 26 cm. Transverse diameter heart 14.1. Ratio heart to chest 54%. Rheumatic history.
Diagnosis- Both mitral and aortic stenosis and regurgitation.
E.K.- Shows no evidence of myocardial involvement.
Fluorimeter- V.C. Markedly pathological. S- Pathological. S+ Normal
T20 Pathological. T25 Subnormal, border line of pathological.

This girl cooperated well. The whole test gives distinct evidence of myocardial lack of tone under strain in spite of the good S+, which may have been the result of some myocardial irritability.

Examiner Residence Date Hour



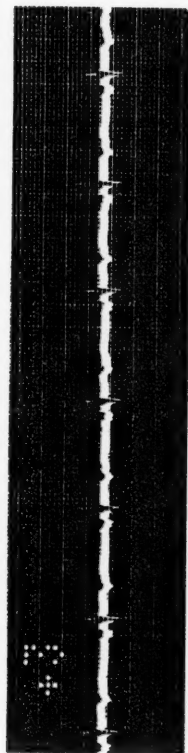
90 S.S. 4-14-21



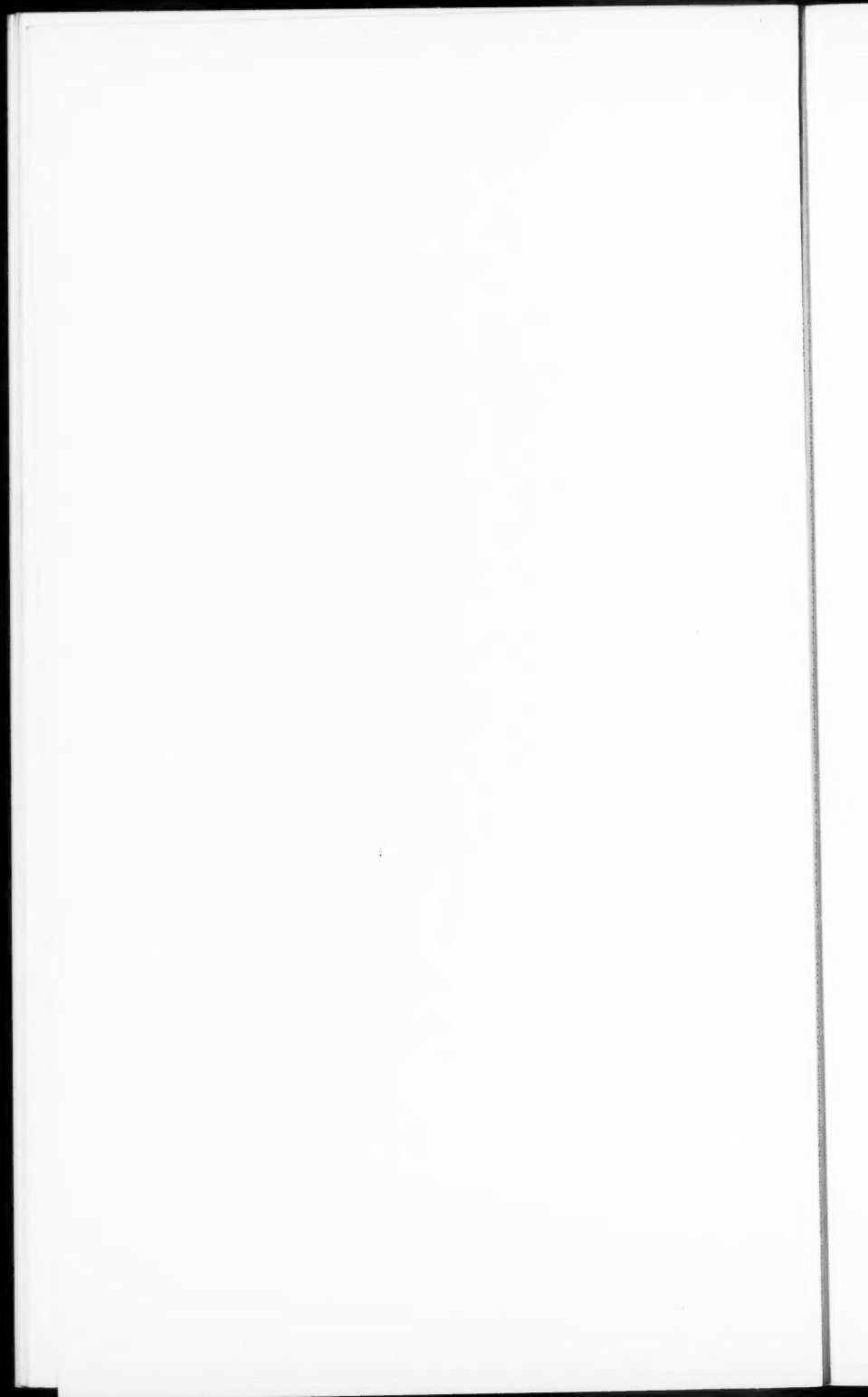
L.1



L.2



L.3



Synopsis of Technique for Functional Tests

Test I	Tests III, IV	Test V	Test VIII
Pulse Rate	Seconds	Seconds	Seconds
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60 Standard Exercise
Diastolic Pressure	Deflate Record	Record Remove Cuff	60-50)
(4th Point)	55-60 Inflate "Inspire!"		60 "Blow!"
	60 "Blow!"	Tests VI, VII	60-60 Heart Rate (per 5 sec.)
	60-20 Systolic Drop to Minimum	30-55 Heart Rate (per 5 sec.)	60-60 Heart Rate (per 5 sec.)
Test II	Deflate Record	Record every 5 seconds	Record every 5 seconds
	25-15 Inflate—T20 in seconds	60 "Blow!"	Underline End of Blow
Vital Capacity	Maximum Systolic	60-60 Heart Rate (per 5 sec.)	
Seconds $\times 0.2 =$ Liters	Total Length of Blow	Record every 5 seconds	
Repeat	Repeat	Underline End of Blow	Continue counting till original rate is reached

RECORD OF TESTS

Name **B. Z. C. 7** Age **58** Sex **M** Occupation **...** Ht. **5** ft. **3** in. Wt. **145** lbs.

I		III		IV	V	VI		VII	VIII
At Rest		Systolic Response		Flameter	At Rest	Heart Rate Response—Flameter		After	Number
						Seconds	Before	During and After Blow	During and After Blow
Pulse Rate	116					0-5	7	7	8
Systolic	158	Systolic	160	156	Systolic	-10	7	8	10
Diastolic	96	Minimum	150	142	Diastolic	-15	7	8	9
		Systolic				-20	7	8	8
		T20				-25	7	8	9
		Seconds				-30	8	9	9
Vital Capacity		Maximum				-35	8	8	8
Sec. Liters		Systolic	170	164		-40	8	9	8
1	13	Total	28	23		-45	8	9	8
2	7	Seconds				-50	8	7	8
						-55	7	7	8
						-60	7	8	8
						Length of Blow	28	Sec.	23
								Sec.	

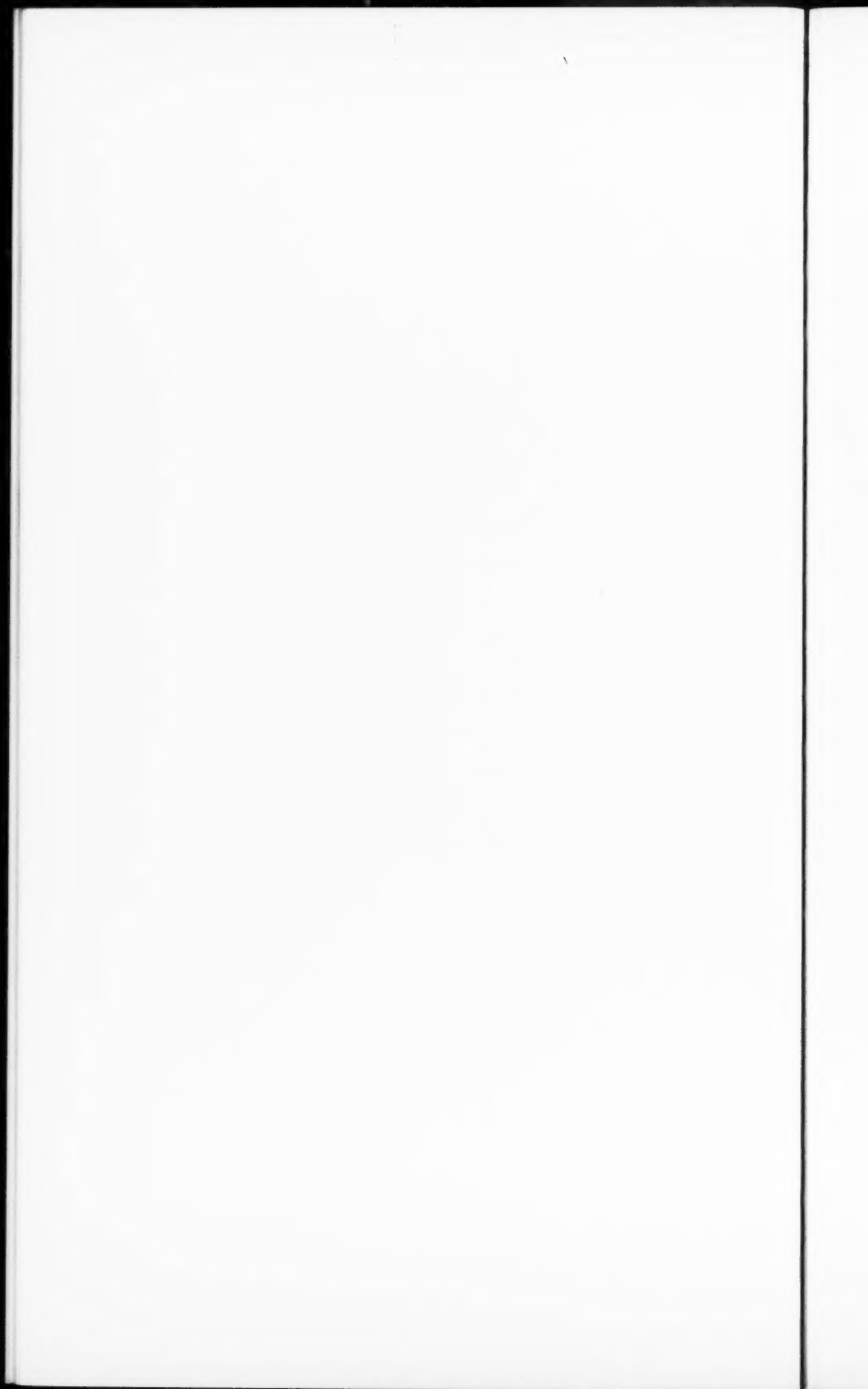
Remarks: **Dyspnea, fatigue, swollen left leg.**
Heart condition first noted in 1928.
Pulse totally irregular.
Diagnosis—auricular fibrillation.

E.K.—Auricular fibrillation.

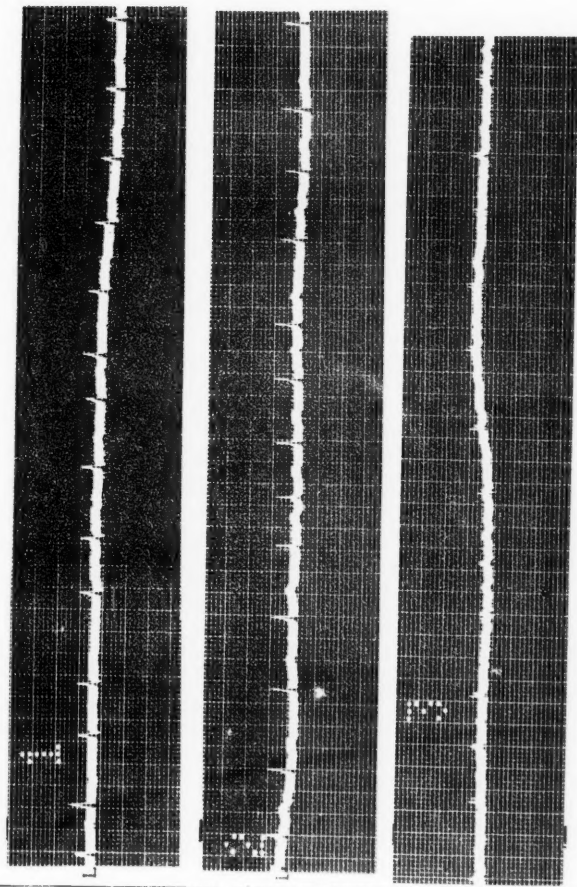
Flameter—V.C. Normal. S—Subnormal. S+ Pathological. T20 Absent.
Tm Pathological.

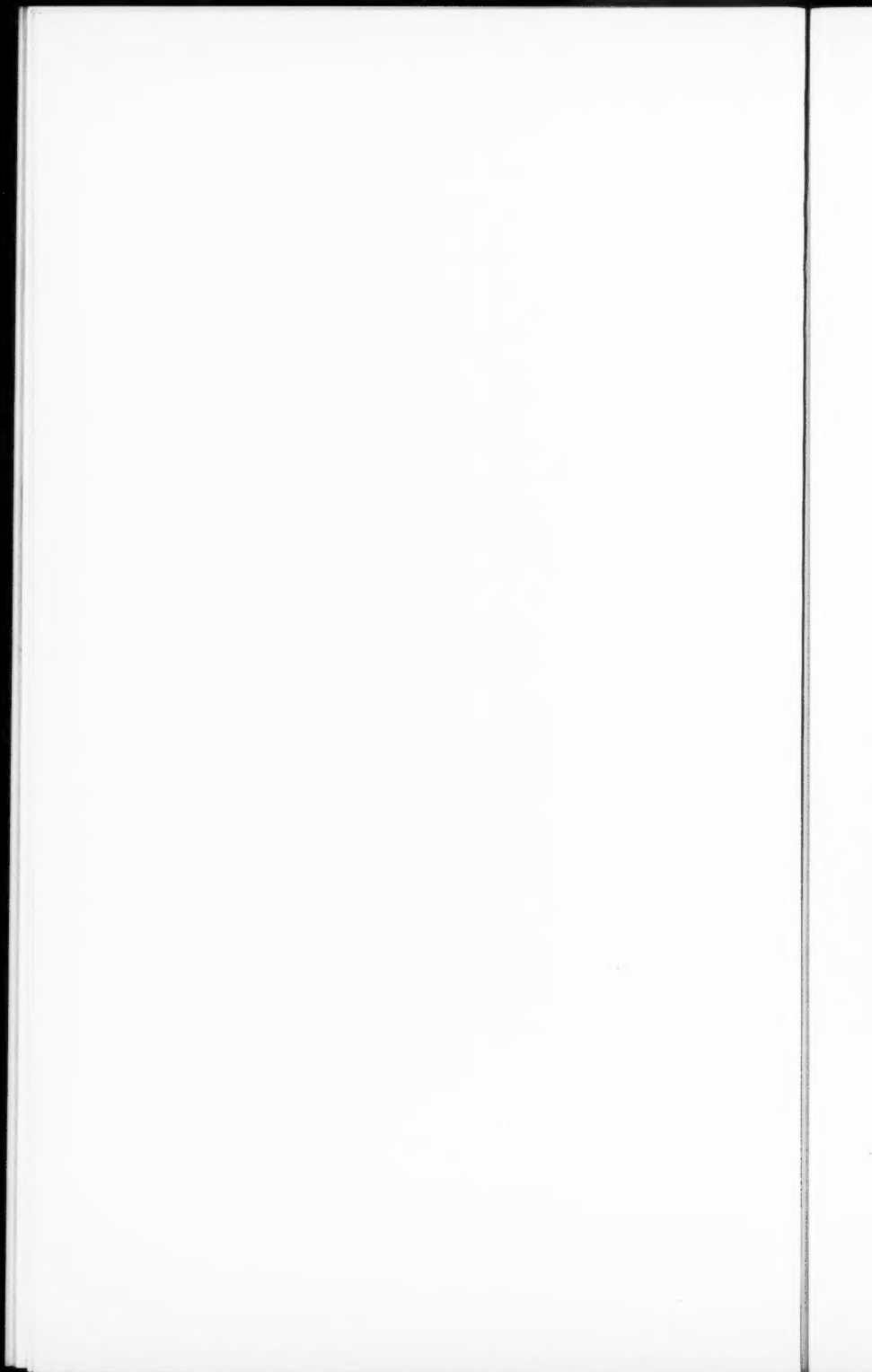
Test points to marked impairment of heart.

Examiner **...** Residence **...** Date **8/12/29** Hour **8.45** P.M.
9900



47 B.Z. 8-13-29 8PM





Synopsis of Technique for Functional Tests

Test I	Tests III, IV	Test V	Test VIII
Pulse Rate	Seconds	Seconds	Seconds
Systolic Pressure	25-45 Systolic Pressure	30-60 Systolic and Diastolic	20-60 Standard Exercise
Diastolic Pressure	Deflate Record	Record Remove Cuff	60-30
(4th Point)	55-60 Inflate "Inspire!"	Tests VI, VII	60 "Blow!"
	60 "Blow!"	30-55 Heart Rate (per 5 sec.)	60-60 Heart Rate (per 5 sec.)
	60-20 Systolic Drop to Minimum	Record every 5 seconds	Record every 5 seconds
	Deflate Record	60 "Blow!"	Underline End of Blow
Test II	25-15 Inflate—T20 in seconds	60-60 Heart Rate (per 5 sec.)	Continue counting till original
Vital Capacity	Maximum Systolic	Record every 5 seconds	rate is reached
Seconds $\times 0.2$ = Liters	Total Length of Blow	Underline End of Blow	
Repeat	Repeat	Repeat	

RECORD OF TESTS

Name R. Z. Age 38 Sex M. Occupation Physician Ht. 5 ft. 8 ins. Wt. 150 lbs.

I		III		IV		V		VI		VII		VIII			
At Rest		Systolic Response-Flameter		At Rest		Heart Rate Response - Flameter		During and After Blow		During and After Blow		Number After 24 Ascents During and After Blow			
						Seconds		Before		After					
Pulse Rate	104	Systolic	132	136	Systolic	140	0-5	4	8	7	6	7	5	7	
Systolic	156	Minimum	114	160	Diastolic	50	-10	7	6	7	6	10	8	7	
Diastolic	46	Systolic					-15	7	5	8	6	10	7	8	
		Seconds	22	18			-20	7	6	8	5	9	8	7	
II		Maximum					-25	7	6	7	6	10	8	7	
Vital Capacity		Sec.	188	200			-30	7	6	8	6	9	7	7	
21.2	4.2	Total					-40	8	6	9	6	8	7	7	
22.0	4.4	Seconds	50	48			-45	9	6	10	6	7	6	7	
							-50	6	9	6	10	6	8	6	7
							-55	7	10	6	7	5	7	7	7
							-60	-	8	-	7	7	8	8	7
% of Normal/Vital Capacity	108	8+	56	64			Length of	50	Sec.	43	Sec.	17	Sec.		

Remarks: Apex beat 1 1/2 inches outside midclavicular line. Pneumonia and empyema. Thoracotomy at 6 years of age. Articular rheumatism when 18 years old.

E.E. - Q.R.S. \pm 0.10 Notched. T inverted-myocardial change. Left ventricular preponderance.

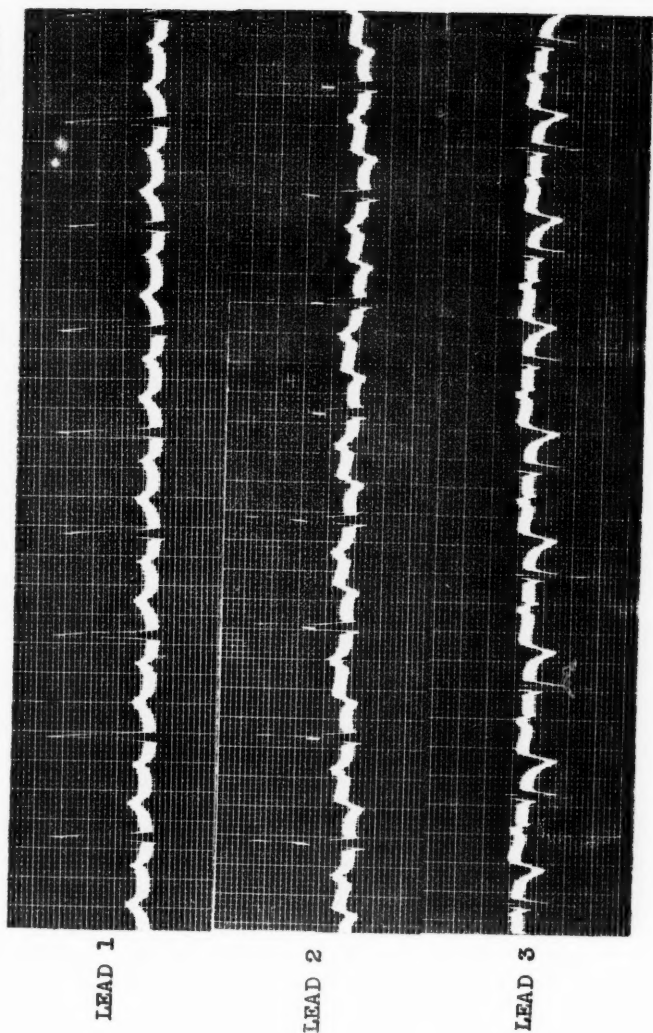
Dr. Polevski

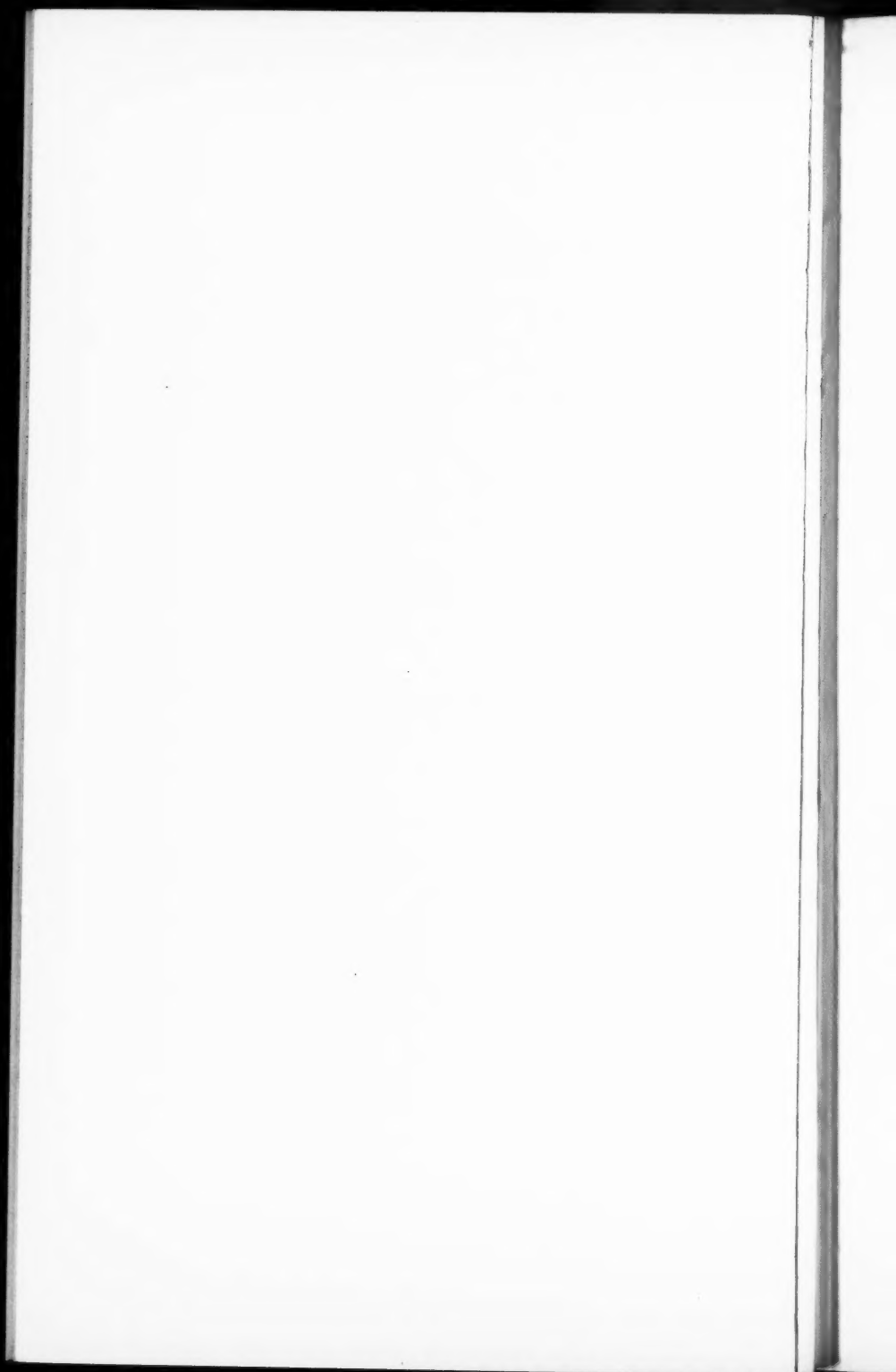
Flameter-V.C. high. S- marked. S+ hypernormal. T20 subnormal. Tn. normal. Attention is called to reported Master's test. It will be noted that while the Flameter blows were 50 and 45 seconds, respectively, before the exercise, the blow immediately following was only 17 seconds, and that the pulse rate was slow in returning to the rate noted at the end of three minutes (84); also that at the beginning of the second minute there was a drop to 60, followed by an instantaneous rise to 96 and after that a more gradual, though variable, slowing, but at the end of two minutes it had only reached 96. This case was referred to us by Dr. Kaufman particularly because he found the exercise test normal and could not understand it. The doctor is up, actively engaged in his work and denies symptoms. The only evidences of impairment noted in the whole test were the subnormal T20 and the delayed pulse return, with possibly some irritability of the heart indicated by a rather high S+.

Examiner.....Residence.....Date.....Hour.....

AUG. 15, 1929

AUG. 15, 1929
Dr. Zimmerman





101

MEN.

Standard Table of Myers.

WOMEN.

Standard Table of Myers.

	Standing Height (inches).										
Vital Capacity (Liters)	56	58	60	62	64	66	68	70	72	74	Vital Capacity (cu. inches)
0.6	%	%	%	%	%	%	%	%	%	%	
0.8	21	20	20	25	25	24	23	22	22	21	49
1.0	35	34	33	32	31	30	29	28	27	27	61
1.2	42	41	39	38	37	36	35	34	33	32	73
1.4	49	47	46	44	43	42	41	39	38	37	85
1.6	56	54	52	51	49	48	46	45	44	43	98
1.8	63	61	59	57	55	54	52	51	49	48	110
2.0	70	68	66	63	62	60	58	56	55	53	122
2.2	77	75	72	70	68	66	64	62	60	59	134
2.4	84	81	79	76	74	72	69	67	66	64	146
2.6	91	88	85	83	80	78	75	73	71	69	159
2.8	98	95	92	89	86	84	81	79	77	75	171
3.0	105	102	98	95	92	90	87	84	82	80	183
3.2	112	108	105	102	98	96	93	90	88	85	195
3.4	119	115	112	108	105	101	98	96	93	90	207
3.6		122	118	114	111	107	104	101	98	96	220
3.8				121	117	113	110	107	104	101	232
4.0						119	116	112	109	106	244
4.2							121	118	115	112	256
4.4								120	117	114	268
	142	147	152	157	162	168	173	178	183	188	
	Standing Height (cm.)										

MALES.

STANDARD NUMBER OF ASCENTS.

(Adapted from Two-step Exercise Test of Master and Oppenheimer.)
Age in Years.

Weight (lbs.)	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59
50-59	28									
60-69	27	34								
70-79	26	32								
80-89	26	30	35	36	35	34	32	30	29	27
90-99	25	28	33	34	32	31	30	28	27	26
100-109	24	26	30	31	30	29	28	27	25	24
110-119	23	25	28	29	28	27	26	25	24	23
120-129	23	24	27	28	27	26	25	24	23	22
130-139	22	23	25	27	26	25	24	23	22	21
140-149	21	23	24	25	25	24	23	22	21	20
150-159	21	22	23	24	24	23	22	21	20	20
160-169	20	22	22	24	23	22	21	21	20	19
170-179		22	22	23	22	21	21	20	19	18
180-189		21	21	22	21	21	20	19	19	18
190-199			20	21	21	20	20	19	18	18

FEMALES.

Weight (lbs.)	10-14	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59
50-59	28									
60-69	27	32								
70-79	26	30								
80-89	26	28	31	32	31	31	30	29	28	28
90-99	25	27	29	29	28	28	27	26	25	25
100-109	24	25	27	27	26	26	25	24	24	23
110-119	23	24	25	25	24	24	23	23	22	22
120-129	23	23	24	23	23	22	22	21	21	20
130-139	22	22	22	22	22	21	21	20	20	19
140-149	21	21	21	21	20	20	19	19	19	18
150-159	21	21	21	20	19	19	18	18	18	17
160-169	20	20	20	20	19	18	18	17	17	16
170-179		19	19	19	17	17	17	17	16	16
180-189			19	18	17	17	16	16	16	15

DIRECTIONS FOR APPLYING THE FUNCTIONAL TEST.

The applicant should be seated in front of the desk with the bared left arm resting on the left slide of the desk. Clothing on the arm will give distorted readings.

Place the Flarimeter in front of him at a convenient distance so that he will be able to use the tube without discomfort or danger of tipping the instrument. Open the box, place glass tube in the socket and lower all valve arms to the horizontal position. Now apply the cuff to the arm, attach the stethoscope directly below it, over the brachial artery, just above the bend of the elbow and a little to the

Functional Tests of the Circulation 103

inner side. It is convenient to have a stethoscope with a ribbon attachment to hold the diaphragm in place, as this permits freedom of both hands. The diaphragm should not be placed beneath the cuff. Attach the tube of the armlet to the instrument when a mercury manometer is used (and this type is preferable). The applicant and instruments are now in position.

Before proceeding with test there are some very definite facts connected with its application which must always be borne in mind. The instrument has two orifices which differ in size, and the force of blow necessary with the large is greater than with the small. The necessity of instructing the applicant how to blow through each is therefore obvious. The large measures the vital capacity; the small breath-holding ability.

The applicant should be seated erect when performing these tests, should take the deepest inspiration he can, and then blow as long as possible, keeping the water at the index level. While he is inhaling the time of the longest inhalation should be noted. The time required by different persons varies from two to seven seconds. The signal "INSPIRE!" should be given the exact number of seconds required by the applicant to complete inhalation before the signal "BLOW!" This is important, because when inhalation is cut short the true vital capacity is not obtained. On the other hand, when inspiration is begun too soon a period of waiting for the signal to blow will occur, and this will again give values which are too low. Having learned the time necessary for inhalation ask him to blow into the large orifice, raising the water to the level of the mark on the tube as quickly as possible, and hold it as near that mark as he can. In this way he will get an idea as to the force of the blow necessary. Before beginning the tests with the small orifice, he should also try blowing in order to learn the force necessary.

When determining vital capacity the signal "BLOW!" may be given at any five-second point on the second dial of

the watch. Supposing that his time of inspiration is four seconds and that the second hand is between the 50 and 55 marks on the dial, as soon as the second hand reaches 56 give the signal "INSPIRE!" and at 60 "BLOW!"—or should the second hand be between 10 and 20 the signal to inspire would be given at 16, and to blow at 20. By using this method it is a very simple matter to note for how many seconds he has blown.

Any blow under 16 seconds in adult males or 12 seconds in females following full inspiration is real evidence of either perfunctory performance or physical defect (See table below).

Normal Lengths of Blow with LARGE ORIFICE by Height—
Adult Males.

Height Length of Blow (Seconds)	(Inches) 56-59	60-63	64-67	68-71	72-75
Normal	18	20	21	22	24
Lower Normal Limit	16	17	18	19	20

The lower limit for females is one-fifth (3-4 sec.) less than for males.

Any blow shorter than the lower limits, shown above, for height will give a Vital Capacity less than 85 per cent. of the normal.

A short blow may be due to improper method of breathing or to cardiac or pulmonary defect.

Improper method must be carefully excluded by urging the subject to inspire as deeply as possible not only by depressing the diaphragm but also by inflating the upper thorax to the maximum.

Many, perhaps most, people do not know how to fully inflate their lungs. Careful instruction, appeal to pride, and real urging to do better is therefore most vital if absolutely misleading results are to be avoided.

We cannot emphasize the need of proper and sufficient inspiratory and expiratory effort too strongly, as it applies not only to the large but with equal force to the SMALL ORIFICE.

Functional Tests of the Circulation 105

The length of blow in seconds with the SMALL ORIFICE should be at least three times the length of the lower limits, by height, shown above for the LARGE ORIFICE. Shortened blows with the SMALL ORIFICE are probably more significant than with the LARGE ORIFICE, and again suggest perfunctory performance or physical defect.

The time taken for the blow varies a good deal not only in different individuals but also in the same person. Its length depends on several factors—the willingness of the subject to cooperate—the depth of the inspiration taken—the physical condition—the will power exerted and the attitude inspired by the examiner. The success of the test will be in direct proportion to the latter. Many subjects doing the test poorly will show marked improvement under sufficient explanation, persuasion and encouragement.

The maximum length of the blow in seconds from the beginning should always be noted and recorded. (THE BLOW SHOULD BE STOPPED WHEN THE SYSTOLIC REACHES 200MM. OR EVEN BEFORE IF THE SUBJECT'S CONDITION MAKES IT ADVISABLE.)

When a blow with the SMALL ORIFICE reaches 75 seconds, stop and record systolic noted at this point. When under 50 seconds use every effort, as suggested, to have the subject inspire more deeply and exert himself to blow longer in the following tests. When the test is completed and the data recorded, pause until the point 25 on the second dial is reached before beginning the next test.

When taking the vital capacity an interval of fifteen to twenty seconds should elapse from the end of the first to the beginning of the second blow.

A time schedule is not necessary in Tests I and II, but they should be completed with the least possible delay as every minute lost adds to the time required for the completion of the tests. We can without difficulty place the instruments in position, give the necessary instructions and complete the series of tests, 1 to 8 inclusive, in less than twenty

minutes and we are sure that a little practice and close attention to detail will enable our examiners to do the same.

Sometimes an applicant when blowing seems to relax his abdominal muscles. This makes it much more difficult to keep the water column steady at the mark. He should be carefully instructed that after taking the deepest breath possible he is to fix his abdominal muscles in order to have as constant an abdominal pressure to blow against as possible, and as the air in the lungs becomes less he can slowly retract these muscles so as to keep the intra-abdominal pressure at about the same degree of tension as the diaphragm rises.

TEST I.

PULSE RATE
SYSTOLIC Pressure
DIASTOLIC Pressure
(4th Point)

Be sure that the applicant is relaxed and composed before observing the pulse rate and blood pressures.

TEST II.

VITAL CAPACITY.
Seconds X 0.2 = Liters
Repeat.

Vital Capacity. Large orifice valve to the right, flow 200 cc. per second. Applicant seated erect. Now have him inspire to the fullest possible extent and then expire as completely as he can through the tube, noting the number of seconds required from beginning to end of blow. This number multiplied by 0.2, will give the number of liters blown. Example 18 sec. X 0.2 = 3.6 liters. Record this and repeat the test.

The interval between Tests II and III is the best time to record the name, age, sex, occupation, height and weight, together with the name of examiner, residence, date and hour.

Functional Tests of the Circulation 107

TESTS III, IV.

Seconds

- 25-45 SYSTOLIC Pressure
Deflate Record
- 55-60 Inflate "INSPIRE!"
- 60 "BLOW!"
- 60-20 Systolic Drop to MINIMUM
Deflate Record
- 25-15 Inflate—T20 in seconds
MAXIMUM Systolic
TOTAL LENGTH OF BLOW
Repeat

Small Orifice, valve to left, flow 36 cc. per second.

Time as a factor now becomes most important. Satisfactory interpretation of the records demands that a definite time schedule be followed. Each test takes exactly two minutes and there should be no delay between them except the pause necessary before beginning the next test on schedule time.

When the second hand on the dial comes to the 25 second mark, inflate the cuff and take the systolic pressure. This can be done rapidly as the approximate height of the systolic has previously been ascertained. As soon as read, immediately deflate the cuff and record the reading. The blow is to begin exactly on the 60 second mark. Give the signal "INSPIRE!" the correct number of seconds (which have already been learned when doing the Vital Capacity test) before the second hand comes to the point 60 on the dial. At 60 give the signal "BLOW!" The cuff should be reinflated, just prior to giving the signal to inspire, sufficiently to raise the systolic pressure to a point at or not more than 5mm. below the systolic just recorded. The systolic pressure falls rapidly and reaches its minimum point usually within ten seconds, but sometimes not until twenty seconds after the blow starts. If it has not been noted by the end of twenty seconds, immediately deflate the cuff but if prior to that time, deflate as soon as obtained. This reading, if caught, should be recorded; if not, put a cross in the appro-

priate space on the form. Under no circumstances should the applicant stop blowing or any attempt be made to find the minimum systolic after the first twenty seconds. Now reinflate the cuff and follow the systolic by quickly raising or lowering the mercury column 2mm. at a time, thus keeping in touch with the pressure until the point T20 is reached.

The symbol T20 stands for the time required from the beginning of the blow for the systolic pressure to reach a point 20 millimeters above the pressure recorded before beginning the blow. See Fig. I. Suppose the pressure recorded was 120mm., then 20mm. above that would be 140. The time required from the beginning of the blow for the pressure to reach 140 will therefore be the time represented by T20.

When the pressure reaches this point the number of seconds should be carefully noted and REMEMBERED. The cuff should not be deflated. Follow the rise in the systolic pressure. When the rise is very rapid the inflation of cuff will also have to be rapid. When the systolic rises more slowly, keeping just ahead of it will be easier. The systolic pressure at the moment the blow stops should be noted and also the length of blow in seconds. Immediately deflate the cuff and record T20, maximum systolic and total seconds to end of blow. Now pause until the second hand again comes to the 25 mark on the dial. (Test IV.) Inflate and repeat Test III.

TEST V.

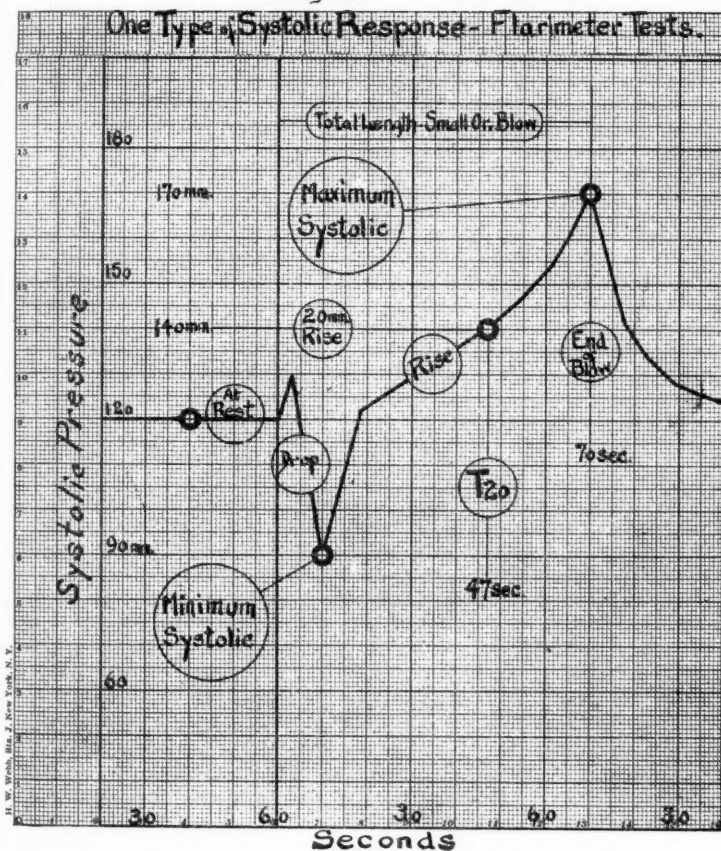
Seconds

30-60 SYSTOLIC and DIASTOLIC

Record Remove Cuff

When the systolic hand again reaches the mark 30, inflate the cuff and record the systolic and diastolic pressures (diastolic again at the fourth point); remove the cuff and record the reading.

Fig. I.



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T
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c

Functional Tests of the Circulation 109

TESTS VI, VII.

Seconds

30-55 HEART RATE (per 5 sec.)

Record every 5 seconds

60 "BLOW!"

60-60 } HEART RATE (per 5 sec.)
60-30 }

Record every 5 seconds

Underline End of Blow

Repeat

In these tests heart rate is substituted for blood pressure readings. Immediately after completion of Test V and removal of the cuff and stethoscope, the latter should be applied to the apex of the heart and its rate carefully counted in 5 second periods, beginning the count each time the second hand reaches a 5 second point. The record therefore starts at 30 sec. as shown on the form and the number of beats in each 5 second period counted from that time on continuously for $4\frac{1}{2}$ minutes, interrupting the counts only when necessary to give the signals to inspire and blow. Each 5 second period missed should be left blank on the form. The Flarimeter is to be again used in just the same way as it was in Test III. As soon as the time arrives, give the signal "INSPIRE!" and promptly at 60 give the signal "BLOW!" Continue counting after the blow stops, but underline the last count recorded during the blow, to help in remembering its length. Record the length of blow in seconds at the foot of the column as soon as convenient, without interrupting the counting. Test VII is a repeat of Test VI.

The reason for taking the heart rate at the apex and not at the radial is that during the blow, particularly in its early stages, pulse volume becomes so small that it may be quite impossible to count it at the wrist.

TEST VIII.

Seconds

20-60}	STANDARD EXERCISE
60-50}	

60 "BLOW!"

60-60}	HEART RATE (per 5 sec.)
60-60}	

Record every 5 seconds

Underline End of Blow

Continue counting till original
rate is reached

This is the exercise standardized by Dr. A. M. Master of New York, at Cornell University Medical College, and is best described in his own words.

"At a given signal, the exact time being carefully noted, and most conveniently taken at a whole or half minute, he ascends the steps. He descends on the other side, and returns, ascending the steps again and descending to his original starting point. This is repeated until his allotted number of ascents has been completed in the one and a half minutes. The turn before each ascent should always be made toward the same side of the room, to insure an alternation of turn to right and left. This is to prevent giddiness or vertigo, factors which produce marked changes in pulse and blood pressure. The patient is kept to the correct rate of stair climbing by observation of the time during the exercise, his rate being accelerated or retarded as may be necessary to complete his quota of ascents in the allotted time."

Tables I, II, adapted from Master's tables, show the standard number of ascents by sex, age, and weight. Find column corresponding to age and follow down to the weight row, where the number of ascents to be used is given. The rate per 10 seconds is slightly more than one-tenth of the number of ascents. For example, 25 ascents in 90 seconds is 2.8 ascents per 10 seconds.

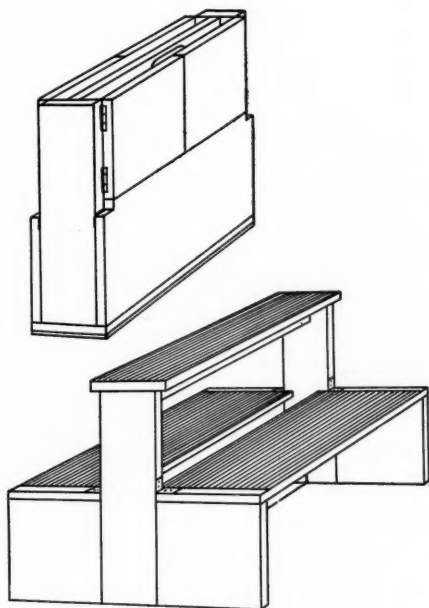


FIG. 2
PORTABLE STEPS FOR STANDARD
EXERCISE.

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Functional Tests of the Circulation 111

The number of ascents made by the applicant are to be recorded in the space set apart on the form between the words "after" and "ascents." Immediately following this exercise, the applicant is to be seated, the stethoscope applied to the apex, and the Flarimeter used exactly as in Test VI. The heart rate is counted by 5 second periods and the counting and recording continued for two minutes. If it does not return by this time to rate at rest, continue the counting for a third minute. The Flarimeter blow after exercise should begin exactly on 60, so lose no time in seating the applicant. It will be noted that this blow is very much shortened. Underline the end of the blow during the count, and record at the bottom of the column the length of blow.

This completes the test. Should there be any unusual features connected with it, especially if the time schedule could not be followed exactly, comments are requested under the head of "Remarks," in order that the test may be properly interpreted.

The orifices of the instrument should never be tampered with, as they have been very accurately constructed.

The water in the bottle must be kept at the proper level. To determine this level, blow through the mouthpiece until the water rises exactly to the index mark on the glass tube. At once close the manometer tube valve. The water should now be at the index levels both on the manometer tube and on the metal tube within the bottle. If below the latter mark, pour water into the mouthpiece little by little to the proper level. If above the mark, pour some out, and readjust as before. It is not necessary to unstopper the bottle. An inspection once a month should be sufficient unless water is spilled by accident.

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DR. PATTON—Dr. Wells will now give us a demonstration or illustration on the use of the test.

DR. WELLS—This is the flarimeter that we have sent out to the examiners. Here is the large orifice and here is the small orifice. We have here a later model, which is slightly smaller and we are now working on the manometer tube. We are not quite satisfied with it, but this represents about what will go into the field.

This is the mouthpiece. The large orifice is with the valve to the right; small orifice, valve to the left. These simply lock when in transit to keep it from leaking.

The vital capacity, as you will remember is determined by taking the longest blow possible through the large orifice with the pressure at 20 mms. of mercury to 172 mms. of water. Here we have a water manometer so that you can follow the systolic pressures roughly. Because of the large mass of water, it lags behind the pressure in the cuff. Here is the laboratory model of the flarimeter and I will use this so that you can see the test. Here we have a set of portable steps.

(Dr. Wells proceeded to give a demonstration of the flarimeter).

DR. PATTON—Owing to the fact that Dr. Rogers has to leave us at 12:30, we are going to ask him to start the discussion of this paper. Dr. Rogers.

DR. ROGERS—I don't know of anything that has been brought before the Association that has interested me more than this device of The Prudential, unless it is the similar work done by the New England Mutual's cardio-respiratory test. I should dislike to say at this time which one of those tests I prefer. It is a little too early, but I like very much the contribution of The Prudential in devising a scheme for measuring the respiratory capacity by means of that carefully graduated orifice. It seems to me that that is a very real contribution, even if the rest of it should, upon analysis, prove not to be so valuable as it seems now likely to be.

From the physiological point of view, from the scientific point of view, I do not feel at all competent to discuss the paper. I don't know enough physiology. I spent my life trying to learn something about life insurance medicine and not physiology. I should prefer to leave the decision as to the value of these tests, the cardio-respiratory and the flarimeter tests, to experts.

But this thought has been borne in on me more and more strongly by this splendid work done by The Prudential: The job is not a job of any one company; I think that it is the job of this Association. The picture that I had in my mind is something like this: You all know the splendid work done by the Committee on Standardizing Urinary Impairments, a work that is continuing and I hope that it will go on year after year by that committee. The picture that I have in mind is a similar picture with regard to this effort to measure the deficiency of the heart. Why should not this work be undertaken by all of us? Why should not a committee of this Association be formed which shall assign, if you please, to each company in proportion to its capacity or ability to undertake the work, to select out a group of lives under conditions that seem fair to the committee, to select out under the cardio-respiratory test a certain number of cases. I know that the company that I represent would go

into work of that kind. Suppose each one of us contributed a proportionate share of observations and kept records of cases so that at any given time, when the actuaries are prepared to help us, we shall be able to lay down 5,000 or 10,000 or 20,000 cases of a very definite sort, measured actuarially to determine the mortalities that have occurred in the groups so separated. It strikes me that ultimately that is what we are bound to come to. It is unfair that any single company should undertake so stupendous a piece of work and be expected to carry it on to its conclusion by itself. We all reap the benefit of it. Why should we not all contribute to it, each according to our capacity to contribute?

That's the picture that I have and I hope that this Association will seriously consider that suggestion—cooperative effort and the avoidance of leaving the burden of proof on any one company or small group of companies.

DR. PATTON—It was with considerable regret that I heard Dr. Rogers speak yesterday of his intention to retire from the more active duties of his office. We are going to miss him, but there is some solace in the fact that his company has not accepted his retirement yet. He is still in an active position at that office, but relieved of a great many of his responsibilities. We are not going to let him go from the Association so far as I can see just yet either, although he may not be at hand to do as much as he has done for and with this Association in the past.

I would like to entertain a motion from somebody on the floor of appreciation of Dr. Rogers' work with us for a great many years, with the hope that he is going to be with us in body as well as in spirit so long as he is able to do so, so long as he is conveniently located. He may be away from time to time. I think some sort of appreciation should be extended by this Association.

DR. McCULLOCH—It seems to me that a valedictory of this type backed up, as we know full well, by long years of painstaking and self-sacrificing service, is worthy of more than some

casual expression we might make extemporaneously at a time such as this. I think that perhaps something more than mere words are due to Dr. Rogers for what he has done for us all, both collectively, and, I can say from my heart, individually as well, with his kindness and his consideration, and I would make a motion, sir, that a committee be appointed to draw up a carefully considered resolution on this subject, and I am in favor of its taking also the form of a material memorial of our regard for him. I make that motion.

The motion was seconded.

DR. PATTON—Are there any further remarks?

The motion was carried.

DR. PATTON—The committee will be appointed later.

We know a good deal about the work of the next speaker and there is little need of an introduction by me. A knowledge of physiology is a foundation that is necessary for the proper evaluation of many phenomena of life that we meet in our work, and I am sure that we will all profit by the discussion that we will now hear from Dr. Yandell Henderson, head of the Laboratory of Applied Physiology, Yale University.

DR. HENDERSON—Mr. President and Members of the Association: I want to express my appreciation of the honor done me in asking me to talk to you.

I am very much impressed with the excellence of the physiology that I have heard in this meeting. A good many years ago I made an address, as chairman of a section of the American Medical Association, urging the importance of clinical physiology. I have been working toward clinical physiology ever since with limited facilities and trying to get other people to do likewise. At last, now, on the 24th day of October, 1929, I find exactly the people to do it. So I wish you God speed. Go right ahead with the sort of work that is being done in this paper. The only thing that I would ask is that, if you get the subject cleared up, you then make your knowledge available to the physiologists. You have facilities for working out new ideas which we have not.

First, there are one or two distinctions that I want to draw. I have heard the expression, *the efficiency of the heart*, many times this morning. I suggest that you need some other expression. The efficiency of the heart is what a man shows who swings a pick-axe or carries bricks up a ladder or who rows on a Yale crew. But I do not think that it what you are really interested in. What you are interested in is something which, for lack of a better term, I would call the "longevity of the heart." You are interested, I take it, in how long the man will live, not, in the case of a man of 50 or 60, how many rounds of golf he can play or how much mechanical work he can do. If the point that you are interested in were really the amount of work that the body can do, I could fit you out right away with reprints of papers and apparatus; and show you how to measure more or less accurately the volume of blood the heart pumps and the amount of oxygen the man can consume. But that is not what you are after. The man might be strong enough to row on the Yale crew, and yet he might die, just in the way that has been mentioned several times this morning, suddenly, a short time later. Many very vigorous men of 40 or 50 die thus suddenly. I have in mind one of the greatest athletes that Yale ever produced who, so far as anybody new, was quite well. I do not know whether he drank too much coffee, or smoked too much, at a meeting he attended one night, but he was found dead in bed the next morning. Now, that was not a case of heart efficiency. He was a powerful man. It was heart longevity, if we can use that term, that he lacked. In my opinion the class of cases which you are aiming at die, as this man probably did, of ventricular fibrillation.

Ventricular fibrillation is a term which has met with great resistance in medicine. A doctor does not mention what he never sees, and he never has a chance to see ventricular fibrillation, because in five or ten seconds after a man has it, he is dead. Ventricular fibrillation cannot be observed like cases of auricular fibrillation. If one says "fibrillation of the heart," the medical man generally thinks of auricular fibrillation, or

complete irregularity of the heart. But ventricular fibrillation is really quite a different thing. Most persons probably have ventricular fibrillation just before death. In carbon monoxide asphyxiation, and in a great many other deaths, the asphyxial process leads to ventricular fibrillation. This can be shown by the electrocardiograph. There are several ways of inducing ventricular fibrillation experimentally. It is quite a definite condition and one which, in my opinion, any heart can go into, but from which the human heart very rarely recovers. You can put a rat's heart or that of any small animal into ventricular fibrillation and it will come out again. Put a dog's heart into ventricular fibrillation and it will quite frequently recover. Put a horse's heart or that of a large animal into ventricular fibrillation and it never comes out, because the size of the heart in relation to the length of the wave of excitation is so large that the wave continues to chase itself round and round. The heart in ventricular fibrillation cannot execute the rhythmic contractions that are necessary to pump blood and maintain life.

The easiest way to induce fibrillation is by the alternating current. When a man dies of electric shock, the ventricle of the heart goes into fibrillation. If you look at a heart in that condition it is "shaking like a bowl full of jelly." Another way to produce it is by simultaneous stimulation of the vagus and accelerator nerves. A third method is by an experiment, first described by Levy, who showed that in cats under excitement a little chloroform will produce it. Chloroform and adrenalin together will also do it. Many a child has died of ventricular fibrillation under light chloroform anesthesia when adrenalin was used in a tonsil operation. In a cat under deep chloroform anesthesia you cannot get fibrillation, but under light anesthesia the injection of a little adrenalin causes the blood pressure to drop like a stone. If you cut the thorax open, the heart is seen to be fibrillating. The animal is dead.

Now, as I said, it is my guess that this is also the cause of sudden deaths in men. I have tried to get some of the great clinicians working on the heart, men like Thomas Lewis of

London, to recognize this occurrence definitely, and to put their authority back of it. I am only a physiologist, but I believe that when a heart shows irregularity without any considerable valvular or myocardial defect, when it shows a tendency to extrasystoles, when the irregularities are increased by smoking, by coffee and by excitement, the patients should be warned that they should stop smoking, that they should not use coffee and that they should avoid excitement.

As yet I have not persuaded any of the leading heart men to take this stand and that is a reason why probably you should reject it and doubt what I am saying. But this I do want to point out in relation to the Flarimeter test: It is not mechanical efficiency, it is not muscular efficiency that needs effective tests; it is the anti-fibrillative capacity of the heart, the capacity of the heart to avoid extrasystoles and not to develop a wave which follows on itself too rapidly. The heart is a pump. It is an engine very much like the engine in an automobile. Like an automobile it has two factors in its activity. It has chambers and valves which produce power, and just as in the automobile engine there is another element, namely ignition, so in the heart there is what you might call the *ignition of the ventricle*. It is the ignition which affords the difficult problem. Defect in the ignition is the factor which causes the bank president to fall dead after a game of golf or the old Yale athlete to be found dead in his bed after attending an exciting meeting of the Inter-collegiate Football Association. The difficulty is to estimate the fibrillative tendency of the heart, for any heart can fibrillate if sufficiently and abnormally stimulated.

With these ideas in the back of my mind, I was much impressed when Dr. Mackenzie and Dr. Wells came to New Haven and gave me a demonstration of this very interesting apparatus. I was impressed by the fact that they lay little stress upon an exercise test. Heretofore heart tests have generally consisted in making the man do a considerable amount of work, and then in determining how efficiently he did it. Exercise tests what may be called the power side of the heart. But it does not

test the ignition side of the heart. The important object is to find a test that will determine the capacity of the heart to counteract fibrillation and to estimate the ignition function of the heart.

Turning to the Flarimeter itself, I think that the test proposed involves three distinct sides, really three distinct tests. One is the vital capacity; this is certainly an extraordinarily clever and useful way of determining the vital capacity. As shown by the late Dr. Francis Peabody, a determination of the vital capacity is a very useful test and measurement. Any patient, any person, with a distinctly decreased vital capacity, unless there is some good reason for it, like an old pleurisy or some trouble of that sort, may be expected to have heart trouble or other disease worth looking into. It is a fairly well known measurement. To measure vital capacity with a watch as the Flarimeter does is a very practical, simple and useful way of doing it.

The second test which is here involved is breath holding. The third is the Valsalva experiment. My attention was first called to breath holding during an expedition to the top of Pike's Peak, where I spent three or four weeks with Dr. Haldane of Oxford. We determined the alveolar carbon dioxide and its converse the volume of breathing for unit mass of carbon dioxide eliminated. We found that after three or four weeks we were breathing a greater volume of air for the same combustion in the body, and that the alveolar carbon dioxide had dropped. At the same time we noticed that the length of time we could hold our breath was shortened. I could hold my breath, as I remember, something like 40 or 50 seconds at Colorado Springs before I went up the Peak, and after three or four weeks at the summit I could only hold it for something like 20 seconds. We watched many people, who came up, go blue for lack of oxygen. Some fainted and we treated them with oxygen. We made many other observations, but the most interesting was this particular observation on ourselves. When Dr. Haldane and I came down the mountain, we both thought we should be able to hold our breath as long as we had before we went up.

But, in fact, at the foot we found we could hold it for only the same length of time that we had at the top; no longer. That observation and many others have shown that the blood alkali, the general acid base balance of the blood, is what is really indicated by the length of time that the breath can be held.

In a student experiment now commonly used, the student finds that he can hold his breath maybe 60 seconds. He finds always that he can hold it longer after forced breathing. I wish we had time for you to join me in that experiment; it requires merely that you do a little deep breathing. After such breathing a healthy man can hold his breath for about three minutes. Then without previous forced breathing the man breathes oxygen. If his circulation is a little bit deficient, he will now hold his breath somewhat longer than without oxygen; but in many cases if his circulation is good he will not hold it longer. The last part of the test is for the man to do forced breathing, and after the period of forced breathing of three, four or five minutes, for him to fill his lungs with oxygen and then hold his breath. He can do this for a surprising length of time. The record for a good many years was nine minutes, and it has been raised now, I believe, to thirteen minutes. Another variation of the experiment is for the man to exercise, and then hold his breath. If the amount of exertion is sufficient to alter the acid base balance of the blood to any considerable extent, the time of breath holding is correspondingly reduced. This morning is the first time that I have seen this test put into practical use and I, for one, got a great deal of pleasure when the subject of the test could hold his breath for only 21 seconds while previously he had held it for something like 60 or 70 seconds.

The third aspect of the Flarimeter test is really Valsalva's experiment. I think that the authors of this test are wise in not pushing the pressure up any higher than they do. There are two illustrations of the risk involved that I can give you. Some years ago, I thought I might be able to develop a method to measure the stroke volume of the heart by blowing into an

apparatus I had devised. I tested it on myself. In my laboratory we generally do the first experiment on a dog, then on a professor and then on a student. (Laughter). This experiment could not be done on the dog, so I started with the professor. I blew into the apparatus fairly hard. Then I blew again, and suddenly I found myself flat on the floor. I had stopped my cerebral circulation and rendered myself unconscious. I did not know when I produced the unconsciousness, but I knew when I picked myself up.

In Yale College, two or three years ago, the officers were considerably worried by the fact that the students were practicing this trick on each other. They asked one of their friends to draw a deep breath and hold it, while they put their arms around his chest and squeezed. The subject always drops on the floor unconscious for a moment, if allowed to do so. Fortunately students do not die easily. However, this experiment shows that it is not wise to push Valsalva's test too far. It is wise to keep the pressure against which the subject blows down to a moderate level.

It certainly is worth while to determine the length of time that the breath can be held, and to determine the vital capacity. There is not, so far as I know, sufficient basis on which to state positively how much value there will be in the measurement of arterial pressure in this test. Yet it certainly ought to be followed up and determined by experience. The initial drop of pressure, then the rise, are interesting, but so far as I can see, their value will have to be shown by statistics. You are in position to find out things that physiologists cannot, for you have immense numbers of cases, each of which is an experiment. All of your subjects die sooner or later, and you have then a mass of statistics out of which you can make inferences on a scale that we poor physiologists who have to use now a dog, now a professor and now a student, cannot hope to attain. To sum up, the tests proposed offer a valuable line of experimentation. In some respects they are absolutely well founded; in other

respects they are very promising. In every respect they are well worth following up. (Applause).

DR. PATTON—The New England Mutual Medical Department has brought much of value to this Association and a few years ago they again made us sit up and take notice with their cardio-respiratory test. The work done by this Company was the real or activating stimulus that led to our investigation. We will now hear from Dr. Frost in the discussion of "Functional Tests of the Circulation". Dr. Frost.

DR. FROST—Mr. President and Gentlemen: It is gratifying to note the extent to which the interest of life insurance medical circles, as exemplified by this association, is being aroused in the desirability, and even the necessity, of discovering satisfactory methods of determining the adequacy of circulatory function.

To clinicians, this need has long been apparent, and to fulfill it they have devoted much of their time and by no means fruitless efforts. While new and more complex laboratory procedures have acquired a well-merited, irreproachable position in the diagnosis of circulatory disease, it yet remains true that the fundamental criterion of the severity of the disease is the degree to which the ability of the circulation to support its load has been decreased.

Longevity and the integrity of the circulation are in direct relation to each other, and apparently to an increasing degree. The latter, therefore, commands the careful attention of the Medical Director. Conceiving the scheme of medical selection with respect to circulatory impairments as an arch, it becomes the keystone.

As to the frequency and the nature of the impairments with which it must be considered, our experience offers some indication. During the period of our use of the cardio-respiratory test,—now over seven years,—we have applied over 9,000 tests to about 8,700 applicants: amounting roughly to 5% of our applications. The nature of the impairments have been heart murmurs, 50.4%; irregular heart action, 7.5%; pulse rate, high

and low, 4%; high blood pressure, 13%; low blood pressure, 2.7%; other circulatory impairments, 4.5%; goitre, 8.7%; overweight, 4.7%; applicants for large amounts and other miscellaneous impairments, 4.5%.

Granting the desirability of testing circulatory function, there are certain practical basic requirements which any technique must fulfill to satisfy the needs of life insurance medicine.

1. It must be reasonably simple. The more simple it is, the more easily it may be acquired by the examiner; the more easily comprehended by the subject; the more extensively employed in the field. Simplicity will tend to eliminate, in direct proportion to its degree, the vitiating effect of the personal equation on the part of both examiner and subject.

2. It must impose upon the circulation a definite load, at least under standard conditions; preferably susceptible to measurement; and adaptable to the varying physical characteristics of each subject. Only thus can comparable results be obtained.

3. It must evoke a definite, readily-measured response from the normal circulation, thus making possible the detection of the abnormal reaction.

4. It must be sufficiently sensitive to effect a definite registration of the minor degrees, of the earlier stages of impaired circulatory function. Advanced circulatory disease, with its major signs and symptoms, does not in general arouse our concern. Its evidence is apparent; its contraindication to life insurance conceded. Its victims constitute but a small proportion of our applicants. There is another considerable proportion, however, which presents the varying signs of circulatory impairment, but lacks definite, relative symptoms. In this group the functional test must play its chief role. Hence the need for sensitivity.

5. It must not embarrass the subject physically or mentally. Otherwise it will defeat its purpose. Preferably, it should capture his interest and concentrate his attention otherwise than upon himself.

6. It must not require an undue amount of time. As to this time element, the sentiment expressed in the paper is, in the main, correct. The question of time may be over-emphasized. A sensible bona-fide applicant,

appreciating that he has a circulatory impairment and that any refinement of examination will militate to his advantage, will not begrudge the extra time involved. Our experience bears this out. Nevertheless, there is an approximate limit which for practical purposes should not be exceeded, having in mind the reactions of both examiner and subject: a period of twenty minutes should suffice.

7. It should not involve an excessive expense, either for apparatus or for the services of the examiner.

Coming more directly to the discussion of this paper, I wish to express my very keen appreciation of the work of Dr. MacKenzie and his collaborators. Their thoroughness in the treatment of their subject cannot be denied. They are to be commended for their careful study of relative literature; for their painstaking designing and calibration of the Flarimeter; for their development and experimental application of the Flarimeter Test. I consider this a splendid piece of work.

The technique of the Flarimeter Test, as at present used by the Prudential examiners in the field, has three essential elements: First, the vital capacity; second, the ability to hold the breath; third, the effect of a standard amount of exercise upon the ability to hold the breath and upon the heart rate. I understand that, as applied at the Prudential Home Office, a fourth factor is being investigated: the fluctuation of systolic blood pressure during the holding of the breath.

While my experience with the Flarimeter Test is necessarily meager, I have, however, applied it enough, to myself and others, to satisfy myself that its technique fulfills the basic requirements outlined above as well as could be expected at this stage of its development.

It is as simple as could be desired, at least as applied in the field. Its technique should be readily acquired by the examiner, easily carried out by the subject. The introduction of the systolic blood pressure determinations undoubtedly complicates the technique for the examiner, but in the light of our experience not to a prohibitive degree.

It certainly imposes a load upon the circulation; under standard conditions, if the body-bending form of exercise is employed; and approximately measurable in foot-pounds of work if the stair-case exercise is used. One is astonished by the degree to which the ability to hold the breath is diminished by the apparently simple body-bending exercise, even in individuals whose physical condition is better than the average.

The response to it is definite and easily determined. By sufficient volume of experiments, it should be possible to discover the range of the normal response. It will not embarrass the subject, provided the amount of exercise is sensibly regulated. It does not require any undue amount of time. The expense involved is by no means excessive.

It will, undoubtedly, reflect the major degrees of impairment of circulatory function. Whether it will register the minor degrees can be determined only by future experimentation. Upon its ability to do this depends its usefulness. I believe it holds forth a great deal of promise.

I confess to considerable doubt as to the practical value of obtaining the vital capacity of applicants presenting a minor degree of impaired circulatory function. In the presence of definite disease of the lungs and circulatory system, its reduction is conceded. But as already stated, applicants thus diseased offer us no particular problem. As indicated by the authors, vital capacity is limited by the size and flexibility of the thorax. It may be increased by practice without any probable corresponding increase in the degree of circulatory function. Conversely, the degree of circulatory function may be appreciably increased, for instance by vacation out-of-doors, without any striking increase in vital capacity.

While it is possible to determine the average vital capacities for individuals of given builds, the mistake should not be made of postulating the average as the normal. Our experience with blood pressures illustrates this point. The average blood pressures for given ages are fairly well established. However, we are equally convinced that the average must not be considered

the normal for all individuals of a given age. Blood pressure varies in accordance with build, temperament, occupation, habitat, and so forth. This, undoubtedly, holds true for vital capacity. There is a wide range for what may be considered normal vital capacity, even for individuals of similar build, depending upon habitat, habits, occupation, and so forth. Compare the sedentary clerk and the athlete of similar build and age. The vital capacity of the athlete will, ordinarily, exceed that of the clerk. Likewise his degree of circulatory function. Yet the lower vital capacity of the clerk may be normal for his occupation and habits.

I believe that the range in normal vital capacity is so large that dependable results from its determination and comparison with an assumed normal range will not materialize. I do not believe it offers a sufficiently delicate reaction; that its fluctuation will be extensive enough to present a dependable basis for evaluation of altered circulatory function. My observation of vital capacity, as obtained in the cardio-respiratory test, indicates occasional definitely abnormal reduction usually in the presence of gross impairment of function and, in addition, a tendency to progressive decrease in successive blows. (In our technique, the subject blows four times to full capacity.) With respect to the minor degrees of impairment, however, I see no convincing evidence of its value.

The portion of the Flarimeter Test which most arouses my interest is the ability to hold the breath and more particularly after exercise. As to the ability to hold the breath per se, there is no question that in the presence of advanced degrees of impairment of circulatory function this will be definitely reduced. In the presence of minor degrees of impairment, however, I am doubtful as to whether this will offer a sufficiently delicate reaction for our purposes. It is, however, necessary as a preliminary to the observation of the effect of exercise upon the ability to hold the breath. As I see it, this latter procedure offers the greatest promise of fruitful results. I believe it may prove sufficiently sensitive to register the minor degrees of impaired function.

I shall be much interested in the conclusions of the authors, after they have completed their experimentation, upon the value of the observation of systolic blood pressure fluctuations during the holding of the breath. I agree with them that the observation of diastolic blood pressure is too difficult and uncertain, at least under present methods, to hold forth any promise of practical value. Furthermore, it cannot be denied that any functional test involving the observation of blood pressure fluctuations as an element of functional reaction labors under a distinct difficulty: namely, the present inability to evaluate with reasonable accuracy the effect upon the blood pressure of extraneous factors, psychic and otherwise. The authors have commented upon this and properly so. Yet in spite of this difficulty, I believe that the observation of systolic blood pressure fluctuations will prove to be a valuable component of the test. I have an idea that it may become evident in the future that the apparent irritability of the circulation which evidences itself by extreme fluctuations of blood pressure, by temporary hypertension, and by unusual sensitiveness to psychic and other factors, is the forerunner, the early sign of the hypertensive circulatory disease of middle and later life. If this should prove to be true, we cannot afford to minimize its importance or to neglect any means of establishing its exact degree.

The authors are to be commended for their painstaking development of the Flarimeter. Without assuming in any sense the authority of a physicist, it seems to me that they have produced an accurately calibrated spirometer, durable, so compact as to be easily portable, and comparatively inexpensive. One cannot question the value of such a contribution. Of itself, it is well worth their time and labor.

In conclusion, I wish again to express to Dr. MacKenzie and his collaborators my very high appreciation of their splendid piece of work. I believe that their Flarimeter Test promises to be of very definite value as an aid in life insurance medical selection. We of the New England Mutual congratulate them. We are in the most complete sympathy with their objective and

their method of approach. We offer them the fullest degree of co-operation within our power.

DR. PATTON—We have learned to listen to the next speaker with the feeling that something worth our time and attention is going to be told us. None of our membership take a greater interest in the advancement of our branch of life insurance, and we are pleased to call upon Dr. William Muhlberg to continue this discussion.

DR. MUHLBERG—Mr. President and Gentlemen: There has been much written and said in the past few years on the mounting death rate from heart and other degenerative diseases, but I believe if the facts are carefully analyzed, the situation will be found not so alarming as the gross figures would indicate. As a matter of fact, heart deaths per thousand per age and before the age of 40 appear today to be slightly less than they were ten years ago. The death rate becomes alarming only past the age of 65 or 70. At these advanced ages, they are no longer of the same importance to Insurance Companies as at the younger ages. But there is, nevertheless, a crying need for more instruments of precision for diagnosing early heart disease and for evaluating its prognosis when the milder grades of it are discovered in our applicants.

It must not be forgotten that after all, the blood pressure instrument is our most valuable aid in averting early heart losses. Cardiac disease at ages past 50 is mostly of the hypertensive variety; and the efficacy of the sphygmomanometer in weeding out such cases has been abundantly proven through the pioneer work of Dr. Fisher, Dr. Rogers and others.

It may interest you to know that in the experience of my Company, of some two hundred deaths recorded in cases giving a history of high blood pressure, but which we nevertheless accepted some fifteen years ago because they seemed to be otherwise gilt-edge risks, 69% were due to cardio-vascular-renal disease with an average age at death of 55 and an average exposure of six years—representing an average heart death rate of almost five times the normal.

I am sure we would all welcome other diagnostic methods or apparatus and Dr. MacKenzie, I believe, offers us such in his Flarimeter. Since Dr. Frost has demonstrated that during a period of five years after examination, risks selected through a somewhat similar method show a favorable mortality, it is incumbent on us to give these instruments a thorough trial, especially in cases of certain borderline impairments. Dr. MacKenzie makes it clear in his paper that his contribution is distinctly in the line of research, but he hopes that future studies will confirm its value.

It is for this reason that all companies ought to co-operate with him in accumulating data through having certain risks submit to this test. It is not entirely fair that Dr. MacKenzie and Dr. Frost should bear the sole burden of these researches. Our companies are all interested, scientifically and financially, and if eventually their instruments are proven of value, we will all avail ourselves of their methods. It would seem only proper therefore, that we endorse their efforts through our co-operation.

About a month ago, Dr. MacKenzie kindly sent me a Flarimeter for investigation and study. I had the opportunity of testing the instrument for only a few days, because I was anxious to have it tried out in our heart clinics by Dr. Schwartz, the cardiologist in charge of the clinics under the control of the Cincinnati Heart Council.

I was really surprised how quickly one becomes familiar with the technique of the apparatus. After the rationale of its method is once understood, it is easy to operate it within the time limits set for the test; and personally, I found that the only real skill required, outside of taking the systolic and diastolic blood pressures, which all of our examiners now know, or should know, was the recording of the figures and watching the second hand of the watch. I wonder whether a metronome, such as musicians use, might not simplify even that procedure.

There is one point, however, that we should never lose sight of, and this is particularly true for heart disease. The instruments of precision can at times give us most valuable data,

and in some cases this information is really pathognomic, but many incipient heart cases will in spite of the electrocardiograph, the orthodiascope, the X-ray, fluoroscope, and blood pressure, etc., escape detection unless the clinical symptoms and its picture are taken into account.

After all, the degenerative forms of heart disease will usually in their incipency give very little evidence detectable by physical examination; but the subjective symptoms are usually quite convincing—increasing dyspnoea on exertion, twinges of pain about the heart or in the substernum or left shoulder or arm, some swelling of the ankles at night, morning coughs and the sense of bloating after meals. Unfortunately, it is precisely this clinical picture that usually is not portrayed in our insurance examinations, but it is the one the cardiologist finds of great aid in arriving at his diagnosis. In other words, in insurance examinations we must encourage every effort that enables us to diagnose heart disease on objective findings alone, because the subjective symptoms are not given us. It is not improbable that Dr. MacKenzie's Flarimeter will help in this way.

Furthermore, any test which gives accurate information as to the heart efficiency today, may give different findings a few days later—not necessarily because the method is faulty, but because conditions in the heart have changed. This is more likely to be true for a heart already badly damaged; a fresh coronary thrombus, or a sudden dilatation due to overexertion may entirely alter the picture. But in insurance selection, we must look ahead many years. A heart with a slight lesion, even if well-compensated, may today be efficient by every known test, but we have as yet no guarantee that it can hold up as well in the coming years, as does a heart without such a lesion. Only statistical study over a period of years can answer that question.

It occurred to me, since I had only a month's time in which to experiment with the Flarimeter, that I could most usefully devote that time to studying the results obtained in cases of advanced heart trouble. Dr. Schwartz kindly agreed to undertake this study. It must be remembered that the cases we treat in

our Heart Clinics are the very advanced cases—many of these patients die of their heart trouble within a few months and most of them succumb within a year or two. But we hoped to derive some data that would be of interest, particularly since the study of the grossly pathological sometimes elucidates the problems of the normal or the slightly abnormal. Physiology, for instance, has gained valuable information through study of the disease or injury to organs whose functions were obscure.

I will read the letter from Dr. Schwartz.

October 19, 1929.

My dear Dr. Muhlberg:

We have some data on about 25 cases which we have examined with the Flarimeter. The majority of these patients did not cooperate well. Most of the cases presented far-advanced, well defined, cardio-vascular disease, and have been under our observation from 2 to 3 years. Of course, to reach definite conclusions from our observations would be folly and yet we cannot help but believe that the results obtained in this small series of cases indicate that the interval between the work done and the response of the cardio-vascular system is directly proportionate to the degree of heart muscle damage present. The response is the elevation of the Blood Pressure in a given time.

We have data on some eight Arterio-Sclerotic Hypertensive patients, with or without Congestive Failure; four Rheumatic Heart cases; one Hyperthyroidism; one Angina Pectoris; one Aortic Aneurysm; and one apparently normal heart.

Case No. 4, as you will notice, is a man who presents perfectly negative physical findings referable to the cardio-vascular system but his response to work falls into limits manifested by cases with definite heart involvement.

Case No. 11 shows an *Aneurysm of the Aorta*. This case apparently shows a fair response to work.

Case No. 17, *Angina Pectoris*, is a man who has been under our care for some three years and who presents negative physical findings, a normal electrocardiogram and yet certainly has a definite organic heart condition. He elicits a poor response to slight effort. This patient is a fit subject for demonstrating the value of this type of test.

Case No. 6, *Hyperthyroid* patient, demonstrates well the use of this machine. Her heart muscle was fatigued and therefore did not respond very well.

The *Rheumatic Heart* cases in this series are of interest. As you know, we cannot help but feel that rheumatic heart disease should be divided

into two distinct classes. The first class includes the apparently healed, perfectly compensated case that suffers no recurrent inflammatory changes of the endocardium. This type of case, if it carries on within its physiological limits has a life expectancy which may be as long as that of the individual who has no organic heart disease. The second class, the type that has recurrent attacks of rheumatic endocarditis and regardless of the amount of rest and management that this case receives, the prognosis is desperate. The cases tested with this machine, we feel, belong to the second class and all but one of these show almost no response to the added stress put on the heart. This exception is Case No. 16 and shows a good response. He is a young man of 30 years of age, formerly a factory worker, but who is now graduating from a school of architecture. The T20 in his Flarimeter test coincides with our clinical diagnosis.

The next group, the *Arterio-Sclerotic Hypertensive* cases, demonstrates well that in high Blood Pressure cases, the danger is not in the high Blood Pressure per se but in the additional ascension of the Blood Pressure during or after work, which apparently may explain why some Arterio-Sclerotic cases show such moderately low Blood Pressures. A marked elevation of Blood Pressure in these cases with moderately low Blood Pressure would be piling up a pathologic physiological state on a pre-existing pathological structure.

Cases Nos. 3, 5, 7 and 8 are cases that from a clinical point of view offer a desperate prognosis and seem to offer evidence that the prolongation of the response to work is proportional to the amount of actual muscle damage present.

Case No. 12 elicits an enormously elevated Blood Pressure with definite pathology of the conduction system and yet there is a ready response to stress.

Case No. 1 manifests a cardio-vascular difficulty in the form of an arrhythmia but this case responds favorably to work.

In conclusion, allow us to state that there is sufficient positive data obtained from this small series of cases as we have collected to justify the use of this machine in a series of 500 cases which could be done in one year.

May I add whereas the Electrocardiograph discloses damaged heart muscle, a functional test such as this may disclose the presence of physiologically active heart muscle.

I also would like to have you suggest to Dr. MacKenzie that the use of some coloring matter in the fluid will facilitate matters considerably for the patient to maintain definite fluid level during the test.

Respectfully yours,

BERNARD A. SCHWARTZ (Signed)

BAS/CS

CASE RECORDS.

Arterio-Sclerotic Heart Cases

Case No. 3	T 20 is 29
1	17
5	34
7	42
8	
9	36
12	12
14	

Rheumatic Heart Cases

No. 2	T 20 is negative
10	"
13	"
16	27

Hyperthyroidism

No. 6	T 20 is 36
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Angina Pectoris

No. 17	T 20 is 30
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Aortic Aneurysm (Lues)

No. 11	T 20 is 22
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Gumma of Urinary Bladder

No. 4	T 20 is —
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He studied some 25 cases and I sent his full reports on some 17 to Dr. MacKenzie. I think the work was very thoroughly done, but in some cases the results, I must confess are somewhat perplexing. Unquestionably in most of these cases we got the result that was expected; in other words, the instrument demonstrated its value. On the other hand, we had a few rather confusing, perplexing cases where in badly damaged hearts it showed a practically normal reaction. We didn't try the exercise test; we were afraid to. However, Dr. Schwartz is very anxious and perfectly willing to cooperate. We are going to examine 500 cases this coming year and I think perhaps a study of those cases will be interesting. This is just an analysis of 17, which, of course, is all we had a chance to study in the very limited time at our disposal.

DR. PATTON—For a number of years, blood pressure was a mooted question for us. No one did more in the earlier years of these tests, nor has anyone shown a greater willingness to do what he could in our attempts to learn more about the circu-

latory system, than has Dr. Henry Wireman Cook, who will give us the results of his consideration of this subject.

DR. COOK—The hour is so late and the discussion has been so very full that I prefer merely to express my very deep personal appreciation of the value that I myself received from a careful study of Dr. MacKenzie's paper. We have not had any personal experience with the Flarimeter at our office, but I believe that the authors are wise in emphasizing the fact that this is a very early and experimental stage and any conclusions would be entirely premature.

I just want to add one comment on Dr. Henderson's remarks, because they were of very great interest to me. Dr. Henderson cautions us, I think, very wisely against accepting too readily an interpretation of cardiac efficiency under unusual strain in terms of probable increased longevity. I am afraid we are all apt to be intrigued by any sort of mechanical plan or method which would give us a short cut, or help us to an easy solution of this serious problem of medical selection. The Yale athlete mentioned by Dr. Henderson would have passed a better efficiency test or better muscular test than any man in the room probably a very short time before his sudden death. We should keep Dr. Henderson's remarks very clearly in mind and be cautious in placing too great dependence on any mechanical heart test as a criterion of probable expectancy.

DR. PATTON—We will now hear from Dr. MacKenzie. We may have to cut him short if we are not going to cut the luncheon short. If Dr. MacKenzie wants to start in now, we'll hear from him.

DR. MACKENZIE—Mr. President and Gentlemen: As Dr. Patton has suggested, the hour is late.

The subject has been very thoroughly discussed here this morning. I think most of the points that have been brought up have been covered in the paper. I do want, however, to say a few words in appreciation, first, to Dr. Henderson for the manifestation of his interest in our work and coming down here to talk to us. We hardly dared to hope for the honor of having

him come and give us the very enlightening talk that he did this morning. I was glad to have him speak of ventricular fibrillation. He spoke to us in a brief way about it when Dr. Wells and myself visited him in New Haven a few weeks ago. He also spoke of the spark plug action and while we have no sufficient data on which to base any conclusive statements regarding systolic variations, we do think, gentlemen, that the T 20, which is the time required for the pressure to rise 20 mm. above the pressure taken just prior to starting the test will prove of real significance in determining irritability of the heart. I don't think there is much doubt about that. The systolic rise at the end of the blow, we are working on and intend to continue but it is not sufficiently worked out.

I also want to express my appreciation to Dr. Frost for the generosity of his discussion and the masterly way in which he has presented it. We were particularly anxious to have the doctor discuss our test because of the splendid work he has already done and because he was really the stimulus to us to start this work, and I want to say that each one of the men who have been responsible for this paper keenly appreciate the manner in which he has discussed this subject.

Now there isn't time, and I don't think it is necessary to say much—it will take a few minutes to go into the room where luncheon is to be served—but I would, however, like to say something about vital capacity. I do believe vital capacity is of value. I think the literature is teeming with the value of that particular test. I think anything below 85 per cent. is suggestive of trouble. Dr. Frost has referred to the wide variations and he has referred to a clerk and to an athlete. Well take those two men, one with a vital capacity of 5 liters and another who exercises somewhat and has a vital capacity of 3, $3\frac{1}{2}$ or even 4 liters and I am inclined to think, gentlemen, that we will get a much better mortality in 100,000 cases with a vital capacity of 5 liters than in a similar number with 3 to $4\frac{1}{2}$.

There is another point about vital capacity and it is this: It does not correlate with total length of blow, as shown by the

small orifice. I think I am right in that. My recollection is that it does not correlate with it, but it will correlate in some diseases. That is a suggestive point. We are going on studying vital capacity. I think it is the least selective of all the blows.

Then we come to the small orifice. I mentioned T 20. That, I believe, is of real value. I have an idea that the total length of blow (small orifice) will be of real value. Vital capacity did not correlate with it in the normal and standard cases, but it did correlate to some extent in the rated cases and more closely in the rejected cases, and it correlated very well indeed with the cases that we handled in Beth Israel Hospital.

This little set of steps, gentlemen, was designed after a study of Dr. Master's test. We think it is rather convenient. It folds up readily, can be put to one side in the doctor's office and takes up very little room. If he finds it necessary to take it out with him, it is hardly as cumbersome as an ordinary suitcase. I think if any of you want to try it, you may be interested perhaps in looking it over.

But the thing that I am most interested in and the thing that Dr. Frost particularly referred to is the shortening of the blow after exercise. You had a demonstration of it this morning. The subject blew, I think, 70 seconds and later he could only blow 21. Now that means something. It means that there is probability of considerable selectivity in that particular part of the test. Now don't think that we are disregarding the systolic pressure at all. We agree thoroughly with Dr. Frost that the systolic in this test is of value. We do think it is a little bit complicated, perhaps, for a great many of the examiners in the field. We don't think any examiner in the field should have any kind of difficulty in asking a man to blow, sit there, watch him and keep one eye on the watch and the other eye on the tube to see that the water is kept up to the mark. We need some such instrument, gentlemen. It struck me rather forcibly when we looked at those electrocardiograms. The electrocardiograms showed an inverted T wave that was manifestly abnormal. The man had no symptoms, he would have passed an insurance

examination with very little doubt and yet he dropped dead in eight days. I don't believe, with our experience with the flarimeter, that he could have passed that test.

Dr. Muhlberg referred to 17 cases given the test by Dr. Schwartz of Cincinnati, and I wish to express my appreciation to Dr. Muhlberg for the interest that he has taken and also to Dr. Schwartz of the heart clinic in Cincinnati for the extremely valuable material that he sent us to look over. Dr. Muhlberg wrote me that some of the results seemed confusing. Well, they were confusing, gentlemen, because I think Dr. Schwartz based his conclusions on T 20 only, but if he had taken the total length of blow in conjunction with it, there would not have been any confusion at all. Every one of those cases that was sent in from Dr. Schwartz showed positive impairment. There wasn't a question about it. Some were better than others, but they ran along the same line, and if Dr. Schwartz had used the total length of blow along with T 20, there would have been no question at all in his mind about it. We are very grateful indeed to have those records. We are going to study them and then return them to Dr. Schwartz.

I want to say that we appreciated Dr. Rogers coming over here and talking this morning. I also appreciate the suggestion that he made about cooperation. Gentlemen, if we can get 10,000 cases or more within one year with the use of this instrument or Dr. Frost's instrument, if some prefer it, why let's get them, gentlemen, and start a mortality exposure. There is value in these tests. There isn't any question about it. They pick up things that the ordinary test won't show. Dr. Collins Johnson of Grand Rapids—many of you know him—one of the leading physicians in Grand Rapids, was in the office about ten days ago and he told me that he had found that the Flarimeter test was of more value in detecting early impairment of the heart muscle in some cases than was the electrocardiograph or the polygraph. Now that's going pretty far. I was glad to hear him say it because it was some indication of what a man of his type thought of the instrument and its value.

Exton—Photo-Electric Scopometer 139

I think I have said enough, gentlemen. I would like to say a lot more. I haven't nearly covered the subject, but it's time for lunch. I thank you.

DR. PATTON—Gentlemen, we'll dismiss for luncheon, and lunch will be served promptly at 1:30. This afternoon we will convene at 2:30.

AFTERNOON SESSION.

DR. PATTON—Laboratory methods and requirements in life insurance have been growing year by year. Most of us no longer take action in questionable cases upon the urinalysis report alone of the field examiner. Home Office laboratories have been developed by many companies and most of the others have doubtful cases and specimens referred to laboratories which they deem competent. The old qualitative tests for albumin and sugar have been replaced by quantitative tests even for the field examiner in most instances. More definite qualitative and quantitative tests are being used in our laboratories; urine specimens are being obtained after special instructions to applicant and examiner; microscopic methods have been improved; blood examinations in diabetic or glycosuria suspects are of daily occurrence. We are most of us familiar with the New England Mutual's methods, as reported by Dr. Dwight, whose urea and specific gravity requirements were some of the earliest advances over the older urinalysis methods. Dr. Exton has perfected instruments and methods that have been very valuable to us in our work and he brings to us today a new instrumental thought. He also has a very valuable suggestion that the Rate of Excretion is of more value than concentration in our consideration of a case. Dr. Exton will now present his papers.

DR. EXTON—Mr. President and Gentlemen: May I preface my remarks on the papers by saying that the laboratory is on this floor and we have arranged demonstrations for such of you as may be interested. All that you have to do in your odd moments or in between meetings is to walk in and ask for whatever you want, that is, albumin, sugar, urea and creatinine in urine, the

method of determining the specific gravity of a drop of urine, and the blood sugar and protein test. I would particularly refer to the quantitative system of microscopy, and think it will interest you to see the new method of microscopy, which I had the privilege of describing last year. There is no question but what this method is simpler and quicker to perform than existing methods and gives far more accurate results. All of you are invited at any time at your leisure to see these demonstrations.

Now about the papers. You have the galleys so I will not take very much of your time. We have been using the scopometer, which, as you now, measures the color or turbid density of a specimen by means of a wedge. The new instrument, of course, is more complicated in construction but its operation is simpler. All you have to do is put in the specimen and move the wedge until you see a flash and then read the result on the scale. I think there is nothing more to say about that. Those of you who are interested can see it in the laboratory. It is not perfect yet by any means, but it will be shortly, and I think that it is the type of instrument you will all use in the future.

Now with reference to the second paper, the rate of excretion, that is a very simple proposition. There is nothing original about it. It states principles which have long been familiar in other branches of science. I simply call it to your attention. What has interested me most in presenting it to you is the matter of its practicability. Would it be possible in insurance work, for instance, to have an examiner see a man and have him pass his urine, note the time at which it is passed and then come back later and collect the urine and measure the volume passed during the time interval, and then correlate the three variables, the time, quantity of urine and the results of concentration tests such as we are now using? It will give you a better method of comparing the random specimens that you have to deal with. I think that is about all; the rest of it is in the paper.

THE PHOTO-ELECTRIC SCOPOMETER.

WM. G. EXTON, M. D.

Director of Laboratory,

The Prudential Insurance Company of America.

To meet the demands of television and sound pictures, photo-electric cells have been greatly improved.

As is well known, the photo-electric effect quantitatively transforms light into electric current which can be put to different uses or measured by suitable instruments. It is therefore practicable to make the photo-electric cell act as a substitute for the human eye in measuring turbidity or color and the possibilities of such an electric eye, as it may be called appropriately, have already been exploited in a number of optical instruments such as Deshler's photometer, Riemann's colorimeter, Sheard and Sanford's hemaglobinometer, and Hardy's recording spectrophotometer.

The several hundred thousand measurements of color and turbidity which have been made with the Junior Scopometer in the course of the routine work of the Prudential Laboratory have abundantly proved its many advantages. Thus, experience has taught us to appreciate especially the new abilities it confers, the simplicities of technic and manipulation it makes possible, and its easy and rapid operation with the great conveniences of enabling measurements to be made without comparison standards and in the same tubes in which tests are done. In use, the reproducibility and accuracy of the Junior Scopometer, as well as its remarkable stability of calibration and freedom from optical and mechanical troubles, have proven more satisfactory than expected.

The principles underlying Scopometry and photo-electric effects naturally suggest that combining them would be ideal in theory. An experimental Photo-Electric Scopometer was therefore put together for the purpose of testing the qualities and possibilities of photo-electric cells and their application to Scopometry. The results obtained were so intrigu-

ing and encouraging that five different models have so far been designed and constructed. The present instrument is thus the sixth. In it are incorporated the lessons taught by experimentation, and in essentials it represents the final design of the instrument.

Although the provisions for electric currents, amplification device, relay and other electrical accessories of photo-electric cells necessarily complicate its construction and give the new instrument an appearance which is somewhat different, the Photo-Electric Scopometer takes even less time and trouble to operate and has all of the practical advantages, such as measuring samples in the same tubes in which tests are made, of the Junior Scopometer. Thus the path of light runs from the source through target, test sample, wedge (or Nicols) and filter in the same order as its does in the Junior Scopometer. Only its termination is different. In the Junior Scopometer the light path ends by stimulating the retina of a human observer and in the new instrument by exciting the electrons of a photo-electric cell. The adaptation field of the Junior is, however, omitted in the new Scopometer because the electric eye does not need it.

The Photo-Electric Scopometer is now being tested and checked against other instruments in order to secure more definite information concerning certain characteristics of photo-electric cells such as stability of calibration, sensitiveness, fatigue, etc. A few months more should suffice to finish these experiments, and I am hoping that by next year's meeting the instrument manufacturers will have made the Photo-Electric Scopometer available to all those interested in its advantages and fascinating possibilities.

THE ADVANTAGES OF RATE OF EXCRETION
OVER CONCENTRATION AS THE CLINICAL
CRITERION IN PROTEINURIA
AND GLYCOSURIA.

WILLIAM G. EXTON, M. D. AND ANTON R. ROSE, PH. D.
The Prudential Insurance Company of America.

It is customary to report the results of urine examinations in terms which denote the mere presence or absence of a given substance (qualitative) or in terms which define the actual amount of the substance in the specimen (quantitative). In previous papers (1), (2), (3), (4), (5) I have pointed out some of the advantages of reporting protein in urine quantitatively, and subsequent experiences in dealing with cases of proteinuria and glycosuria have established the fact that quantitative reports furnish much more concrete and definite information than qualitative reports. It appears that clinicians do not universally require quantitative reports because they do not yet appreciate their value, a circumstance which may be explained by reasons which involve both the technics of urinalysis and the difficulties of interpreting the clinical significance of urinary findings.

With regard to the technics of urinalysis, it may be said that determinations of protein and sugar in urine can now be made with a precision satisfactory for every clinical purpose and with negligibly more time and trouble than it takes to make the qualitative tests in common use. With regard to interpreting the clinical significance of urinary findings much greater difficulties confront clinicians. It is, therefore, our present purpose to call attention to and illustrate certain principles which underlie the clinical interpretation of findings of albumin and sugar in urine, principles which have long been familiar to scientists, although they are scarcely mentioned in the literature of clinical medicine, perhaps on account of their elementary character.

The perplexities which bother clinicians when they attempt to interpret the significance of albumin and sugar findings in a patient's urine are often fundamental in origin because they arise from the discrepancy existing between the information about elimination which clinicians need and expect to derive from urinalysis and the information given by our laboratory reports. Thus, in practice the urine is almost always examined with the purpose of obtaining information about the patient's elimination or excretion of albumin or sugar, while our laboratory reports tell only their concentration. Concentration being only one of the several factors included in the concept of elimination or excretion, it follows that our laboratory reports do not provide the kind of information that clinicians need.

The clinical concept of concentration is limited to a measure of the concentrating capacity of the kidneys, especially when the concentrations of a given substance in blood and urine are compared. Thus, the specific gravity of the urine is a function of the concentrating capacity of the kidneys. On the other hand, the clinical concept of elimination or excretion is much broader, and comprehends:

- A—A measure of the eliminating capacity of the kidneys, which, of course, includes other factors than concentration.
- B—A measure of the production in the body or the intake into the body or the utilization by the body of substances which must be eliminated or excreted by the kidneys.
- C—A measure of the retention within the blood or body tissues of substances which are not excreted as they should be on account of faulty metabolism, altered function or organic disease.

A clear distinction between concentration and excretion makes it plain that what clinicians really want and need is the best information they can possibly get concerning the elimination and not the concentration of excretory substances which happen to interest them. Such information can easily

Exton-Rose—Proteinuria and Glycosuria 145

be obtained by correlating albumin or sugar concentrations with other variables.

It is well known that the greatest variable in the composition of body excretions is water and that fluctuations in water content profoundly affect the concentrations of excreted substances like protein and sugar. Taking the water into consideration enables us to determine the total elimination of an excretory substance, i. e., its concentration multiplied by volume of urine. While total excretion is often a better clinical guide than mere concentration, our study of cases excreting albumin and sugar shows that the total excretion also fails to give truthful information about elimination unless correlated with a third variable, i. e., time.

The literature gives some data concerning elimination in terms of the total excretion over a twenty-four hour period, and such a clinical criterion represents not only the total mass of the excreted substance but also its rate of excretion. A twenty-four hour period is, however, not always practicable; nor is it sufficiently informatory because the excretion over a short interval of time often has a very different clinical meaning than the same excretion over a long period of time, and it is, of course, well known that the excretion during a small part of a day may have an entirely different significance than the elimination over a twenty-four hour period.

As a unit of elimination, the hourly rate of excretion is therefore suggested as a satisfactory and convenient clinical criterion. To ascertain the hourly rate of excretion it is only necessary to measure the amount of urine passed during any known time interval and apply our albumin or sugar tests as usual.

As a practical matter, note the time at which a patient completely empties the bladder and measure and examine the urine passed at the next later voiding. Then multiply the volume of urine (cc.) by the concentration as shown by quantitative tests, and divide the product by the time in-

terval, whatever it may be. In this way the excretion rate of specimens collected in less than an hour may be determined but longer intervals are preferable and catheterization may be advisable in some cases.

It will have been observed that the rate of excretion is derived from the correlation of three variables: concentration, urine volume and time, and the effects of their interplay between one another on the resultant may be illustrated by the case of a diabetic under treatment. Mr. X's urine showed 1% sugar, and his physician modified his diet and insulin, in accordance with good practice, with a view to reducing his sugar elimination to normal. Two weeks later Mr. X's urine gave only a faint reaction by Benedict's test and no reaction at all with Fehling's. The results of these tests were naturally interpreted by Mr. X's physician as meaning that the changed treatment was effective, but, as a matter of fact, Mr. X was actually excreting three times more sugar at the time his urine appeared to be practically normal than he was excreting when it showed 1%, because he was eliminating sugar at the rate of 166 milligrams per hour when his urine showed 1% and at the rate of 500 milligrams per hour when the concentration of sugar in his urine was only $\frac{1}{4}$ of 1%. In other words, Mr. X's condition was misjudged because concentration proved to be a misleading clinical criterion. On the other hand, had Mr. X's physician reckoned that the 1% specimen represented 40 cc. of urine passed during 140 minutes, and that the practically negative (0.25%) specimen represented 200 cc. of urine passed during 60 minutes, the true state of Mr. X's sugar elimination would have been apparent.

Our protocols show that almost all cases of glycosuria or proteinuria exhibit similar instances at times. Thus in another case a patient whose urine showed 7% sugar at two different times was excreting at one time 327 milligrams per hour and at another 2650 milligrams per hour, and a patient whose urine showed 2% sugar at two different times of the

Exton-Rose—Proteinuria and Glycosuria 147

same day was excreting at one time 980 milligrams per hour and at another 1960 milligrams per hour. Figure 7 shows a similar example.

The following instances, taken from case records, will serve to illustrate the separate effects of each of the three variables entering into the calculation of the rate of excretion:

TABLE I.

Case	Time Hours	Volume cc.	Concentration mg. %	Rate of Sugar Excretion Milligrams per hour.
Constants: Time and Volume Variable: Concentration				
C 109	1	25	3.5	89
	1	32	6.5	208
C 103	2	91	0.4	18
	2	90	1.7	77
Constants: Time and Concentration Variable: Volume				
C 103	1	183	85.0	15725
	1	130	84.0	10900
C 104	2	75	18.0	675
	2	200	15.0	3000
Constants: Concentration and Volume Variable: Time				
C 93	2	240	0.1	12
	1	230	0.2	46
C R	1	110	6.4	704
	4	100	6.4	160

That identical rates of excretion may be the resultants of considerable differences in the variables is evidenced by the following instances from case records:

TABLE II.

Time Hours	Volume cc.	Concentration mg. %	Rate of Sugar Excretion Milligrams per hour.
2	200	19.6	1960
2	110	35.2	1940
8¾	425	11.7	570
3½	440	4.5	577
6	250	9.5	400
3	215	5.4	400
2	50	16.0	400
2	110	7.2	395
3	190	4.9	310
4	425	3.0	310

These data might be multiplied by many other instances, but those given suffice to make it clear that much more accurate information can be secured by considering all of the three variables that enter into elimination than by depending on only one of them, as is our present clinical practice. Cases which run extremely high excretions may be regarded as exceptions because in such cases qualitative tests often meet clinical requirements.

In previous papers I have given methods for portraying elimination by graphs on which are plotted separate curves for specific gravity, urine volume, concentrations and rates of protein or sugar excretion, etc. Measurement and examination of the urine voided every two hours furnish the necessary data which it takes about fifteen minutes to get by new quantitative methods with the Junior Scopometer. Such graphs have proven very interesting and informative because they give complete and truthful pictures of a patient's daily excretion of albumin or sugar, and delineate the effects of digestion, activity, complications, treatment, etc.

The two graphs (Fig. 1) of G's. protein excretion serve for illustration. G's. protein elimination was first typed at a time when he sought relief from malaise, and about a year later when he felt perfectly well. On both occasions the specimens were collected during the course of his day's work as a clerk between 8:30 A. M. and 4:30 P. M. On the 1925 graph it is interesting to note that the concentration and excretion rate curves run parallel with one another. Their trends being the same, both curves give about the same kind of clinical information. Instances like this and the cases having extremely high excretions constitute the only conceivable exceptions to the general rule of excretion rate being a better clinical criterion than concentration.

While concentration and excretion rate curves often tend to run parallel with one another when people are resting during the night, our data indicate that this simple relationship is unusual during more active parts of the day. Thus

the 1924 graph of the same case shows the concentration and excretion rate curves varying in their relationships with one another in about every conceivable way.

Taking into consideration the fact that one of the graphs was made a year later than the other the protein eliminations are remarkably similar and indicate little if any change in G's. condition. It is to be noted that any random sample of urine passed by G. during the active hours of either day shows an excretion rate which furnishes truthful clinical information about his protein elimination. On the other hand, the concentrations are misleading. Thus, the concentrations of protein in specimens passed during the mornings were two to five times higher in 1924 than in 1925, when G. was actually excreting just about the same amount of protein. At noon in 1924 the concentration of protein is at its highest just when G. is excreting less protein than at any other time of his active day, while at noon in 1925 the concentration is lowest, when he is actually excreting more protein than at any other time of the day. Such shifts are due to accidental causes connected with activities, time and nature of meals, etc., but in every instance more truthful information concerning elimination is given by excretion rate than by concentration.

The graphs of G. also illustrate another point bearing on the clinical interpretation of albumin and sugar findings in urine, which is important because it goes to the root of a clinical error which is made much oftener than is commonly supposed. Some of the albumin tests in common use are not sensitive enough to show the presence of protein when the concentration runs 10 mg. per 100 cc. or less, and such tests easily mislead physicians to diagnose as intermittent cases which are in fact persistent albuminurias. In practice such instances often occur and patients not infrequently excrete more protein when the tests appear negative than they do when the concentrations run high enough to give positive qualitative tests.

Glycosuria cases often exhibit a reversal of this situation by passing random specimens containing sugar in high enough concentrations to give positive qualitative tests when the actual excretion of sugar is normal or even less than normal. Such glycosurias are usually benign, but, on the other hand, some glycosuria cases pass urines which test negatively at times when they are excreting more sugar than when the qualitative sugar tests are positive.

Instances like these happen so often and unexpectedly in practice, and particularly in doubtful cases, that they suggest the advisability of revising our clinical definitions of intermittent and constant because the distinction between these terms can be correctly made only when based on the excretion rate calculated from the results of the most sensitive available quantitative tests.

In practice, physicians must usually put up with the random specimens of urine patients furnish when they visit office or clinic, and judge the patient's condition by comparing reports of urinalyses which have been made from time to time. If he relies on concentration alone, the physician is apt to reach false conclusions because he ignores the irregular but often marked effects of volume and time. On the other hand, if his criterion includes these effects as does the rate of excretion, the clinician can always compare urinalysis reports strictly and truthfully.

The cases of P-47 and P-41 (Fig. 2) differ greatly in clinical type and symptomatology. Note that P-41's concentrations of protein ran so high that it was necessary to reduce them five-fold in order to bring them within range of the graph. A comparison of the graphs, however, shows that while P-41's concentrations average almost seven times more than P-47's, he is excreting only about twice as much protein. In other words, in this case true and false criteria of elimination differ by a factor of three. P-47's excretion rate curve is characteristic of a type shown by the graphs in a number of other cases of long standing albuminuria. Note

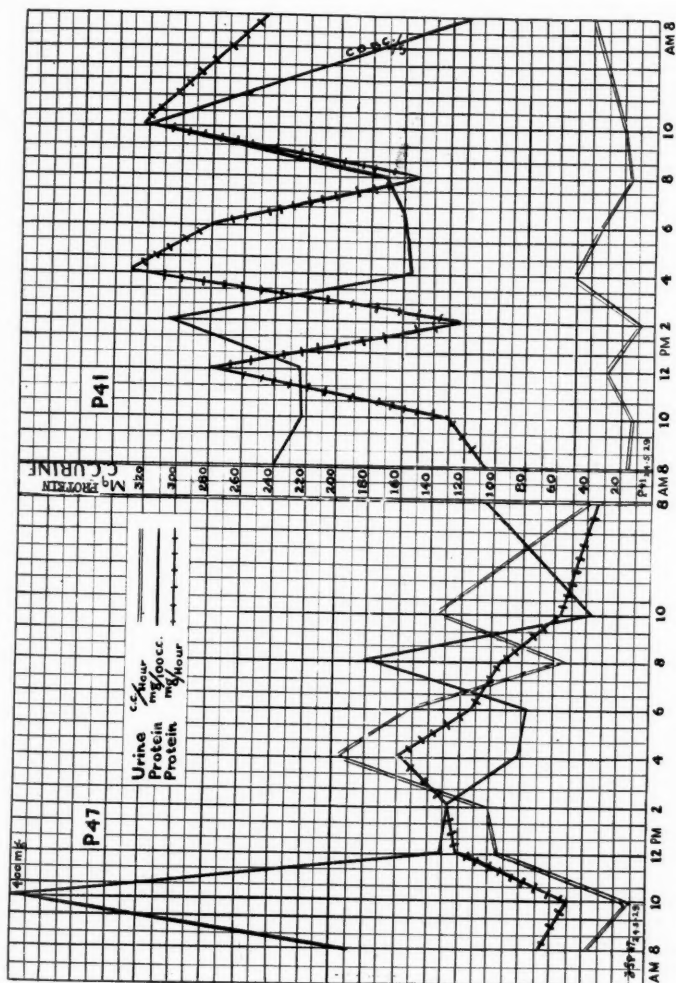


Fig. 2

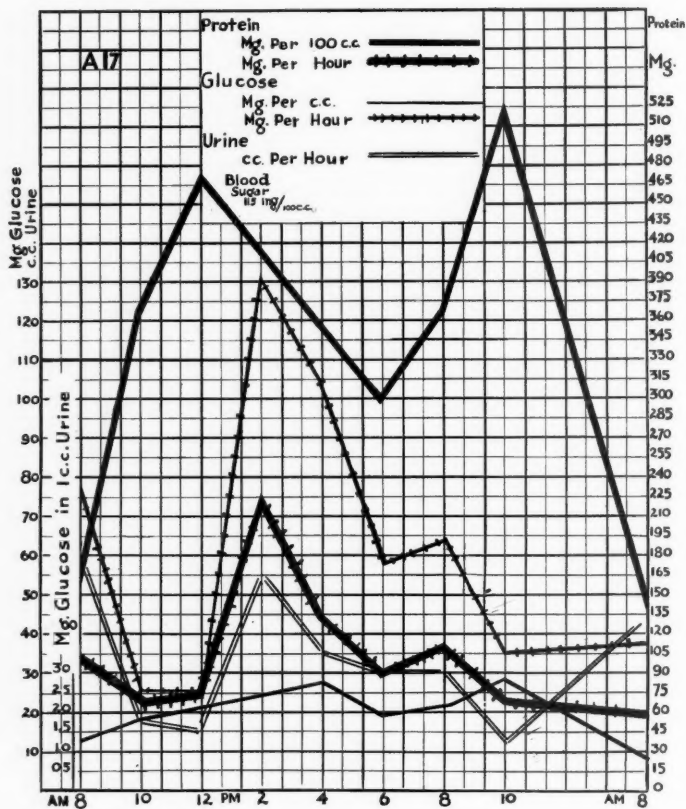


FIG. 3

its gradual rise with the day's activities and its gradual decline with their subsidence, which is in marked contrast with the violent irregularities of P-47's concentration curve.

P-47's concentration curve also illustrates the misleading information given by random samples when concentration alone is employed as the clinical criterion. Incidentally, it may be noted that it is only rarely that a graph does not show one or more such instances. Concentration alone gives misleading clinical inferences depending on the time of day at which specimen happens to be collected. Thus, a specimen collected from P-47 around ten in the morning gives a clinician an impression which is quite different from that given by P-47's late afternoon or even his first morning specimen, which so many physicians prefer for examination.

It will not have escaped observation that P-47's extreme differences in concentrations are largely due to the effects of urine volume. Thus, during the day P-47's protein excretion rates fluctuate over a fivefold, his concentrations over a tenfold, and his urine volume over a hundredfold range. It will likewise be noted that at 10:00 A. M., when concentration is markedly at its highest, P-47 is actually eliminating less protein than at any other time of the day. At 4:00 P. M. a reversal of this situation takes place because P-47's elimination is then at its peak for the day but his concentration of protein is only a fifth of what it was at 10:00 A. M. After the day's activities the irregularities of the concentration curve contrast markedly with the gradual decline of the excretion rate. So many such instances show on our graphs that the clinical advantages of employing a criterion which takes all instead of only one of the variables involved in elimination into consideration seem self-evident.

The graph of A-17 (Fig. 3) shows very simple relations between his protein concentration and excretion rate curves, and it will be observed that their trends are opposite one another instead of parallel, as in G's. (Fig. 1), 1925 graph. In instances like A-17, where concentration and excretion

rate curves trend opposite one another, concentration as the sole criterion tends to exaggerate the degree of albuminuria. When determined by iron reduction, A-17's blood sugar turned out to be higher than normal, and it is interesting to note that his blood sugar and excretion rates indicate a mild diabetic condition which his sugar concentrations fail to suggest. It is also of interest that the excretion rates run parallel while the albumin and sugar concentrations show no such tendency.

A-15 and 16 (Fig. 4) are diabetics. They get the same calories and no longer take insulin. Neither concentrates sugar enough to give positive qualitative sugar tests but it is interesting to find that A-16 is excreting only half as much sugar as A-15.

A-6 (Fig. 5) is a diabetic on 47 units of insulin. In contrast with his hypoglycemia and subnormal concentrations of urinary sugar the fluctuations of his excretion rate curve suggest his real condition and perhaps changes in therapy.

The graph of A-12 (Fig. 6) illustrates the effects of water excretion on sugar elimination and how concentration misleads by ignoring urine volume. At 8 P. M. A-12's sugar concentration is less than normal and below the range of the qualitative sugar tests. At that time, however, A-12 is excreting sugar at an abnormally high rate and passing more urine than at any other time of the day. In such instances excretion rate enables the clinician to diagnose glycosurias which are not shown by the qualitative sugar tests. Two hours later A-12 is passing very little urine, which explains why the excretion rate falls to normal at a time when the sugar concentration reaches 0.8%. Such wide fluctuations in concentration and excretion rate seem to be characteristic of patients getting insulin or perhaps having renal diabetes. On the other hand, normal people with functionally competent kidneys often concentrate sugar and other excretory substances in apparently abnormal amounts when for some reason, like sweating, they eliminate very little water.

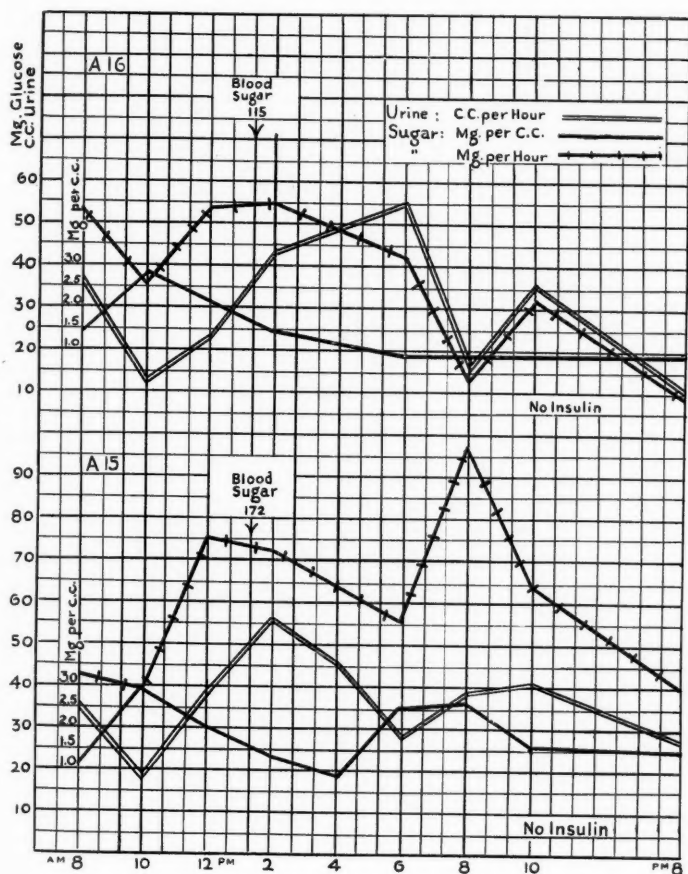
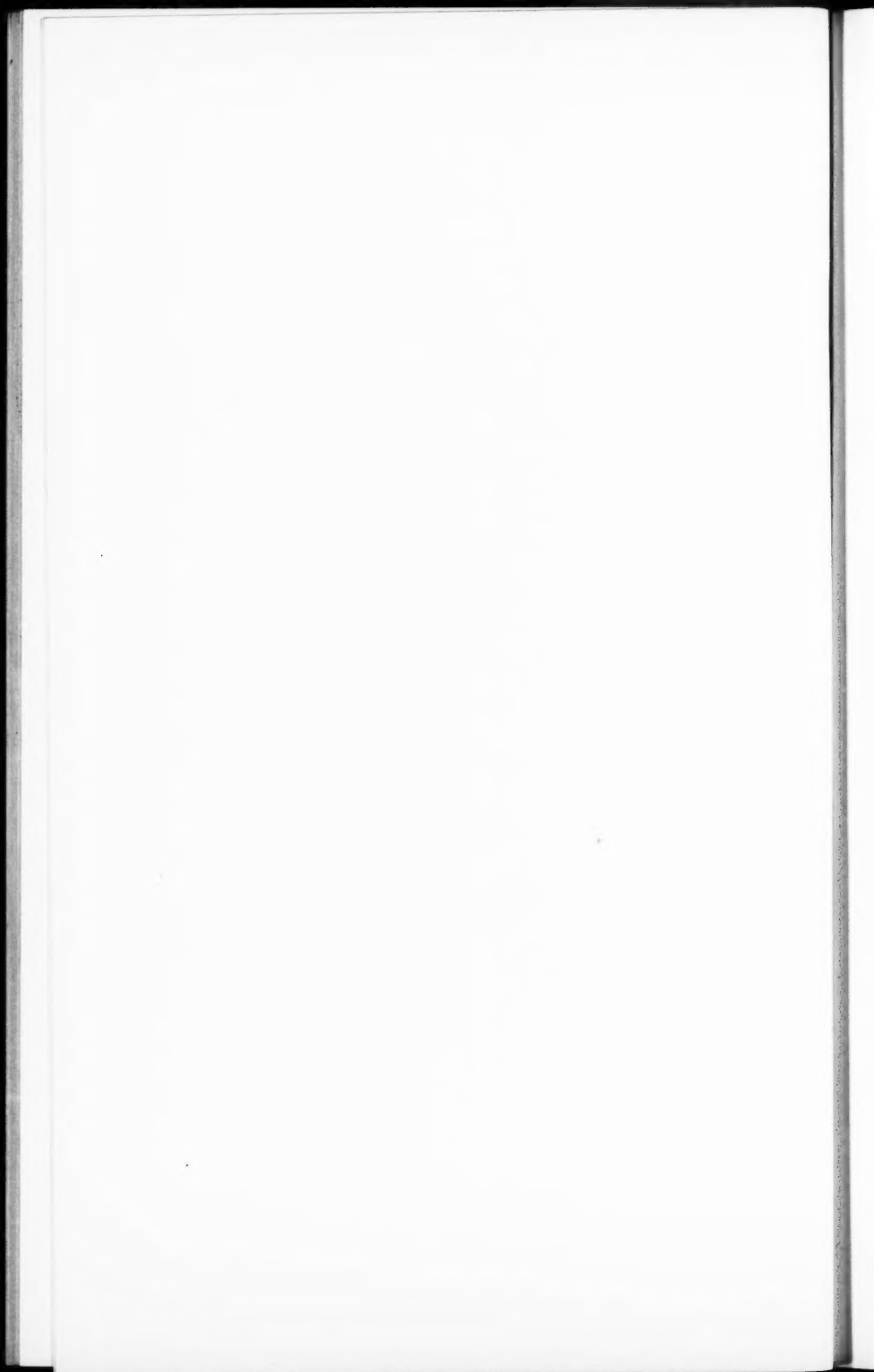


FIG. 4



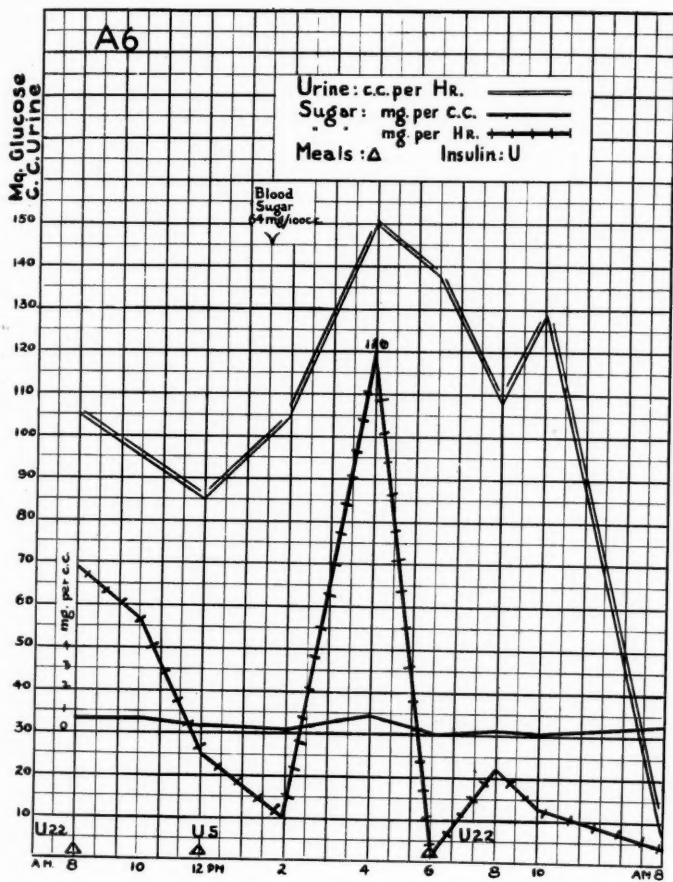
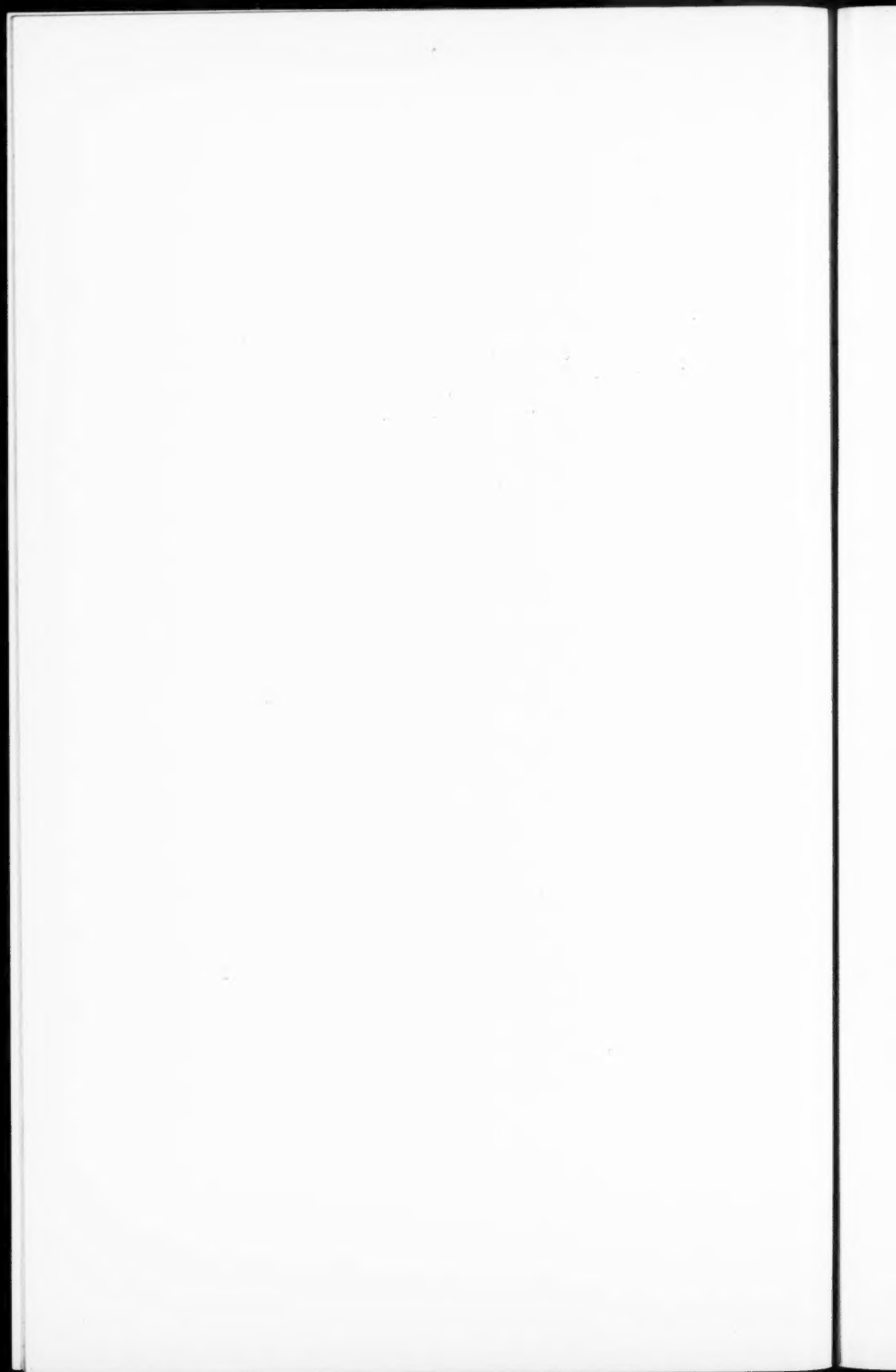


FIG. 5



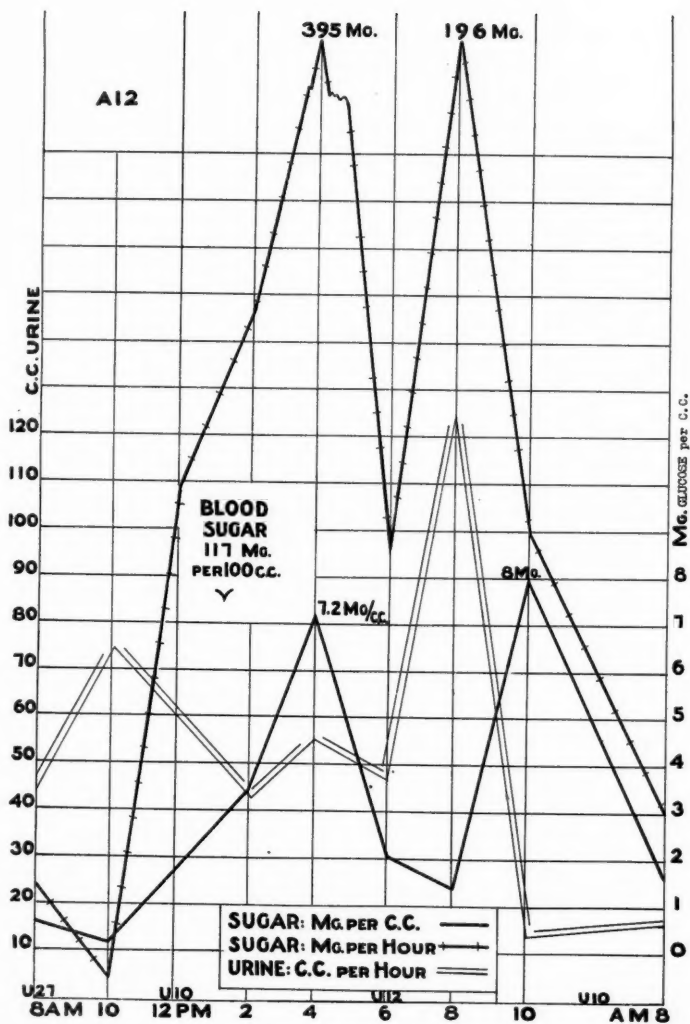


FIG. 6

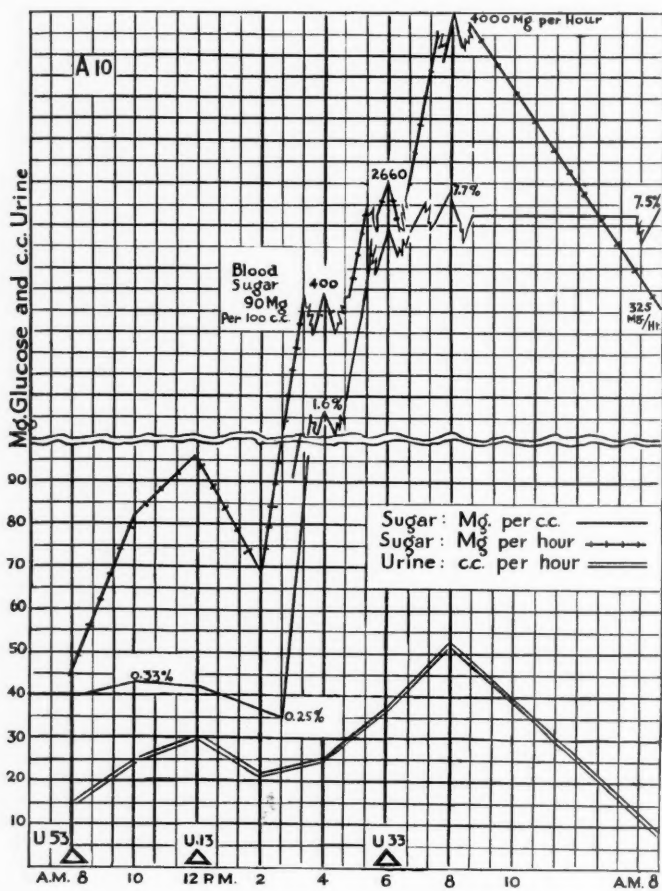
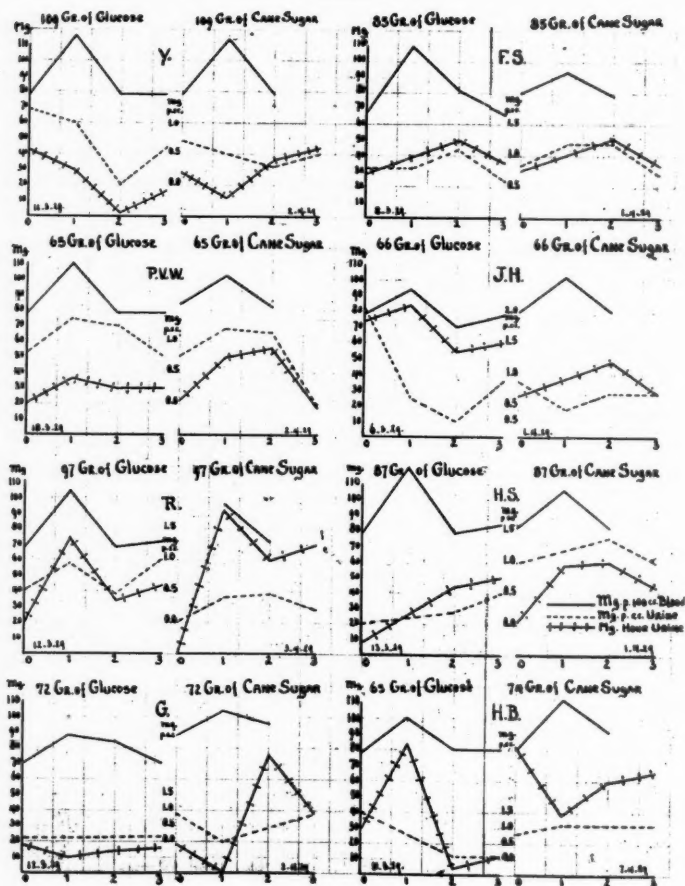


FIG. 7

FIGURE 8.



Exton-Rose—Proteinuria and Glycosuria 153

In such instances excretion rate gives truer and more reliable information about their elimination than does concentration. That high rates of excretion can occur independently of large urine volume is evidenced by points on the curves at 4 P. M.

A-10 (Fig. 7) is a diabetic getting 99 units of insulin a day but not yet under control. His concentrations fluctuate over a thirtyfold and his excretion rates over a hundredfold range—a condition which seems to be characteristic of renal diabetes and insulin therapy. Between 5 and 8 P. M. his concentrations remain constant at about 7.5% but his excretion varies from 325 to 4000 milligrams per hour. This and other graphs suggest the possible usefulness of the excretion rate peaks for timing insulin.

From the standpoint of insurance selection Fig. 8 is particularly interesting. It shows the concentrations of sugar in blood and urine and the sugar excretion rates of eight normal individuals of different ages who were given carbohydrate tolerance tests with both glucose and cane sugar. The results of both tests are placed side by side for convenient comparison and may be regarded as typical of normal between ages 20 and 55 because they are similar to data obtained in a number of other cases.

It will first be noted that the matter of glucose or cane sugar makes little difference in the blood sugar curves. The concentrations of sugar in urine, however, run higher after cane sugar than they do after glucose, especially at the second hour. The rate of sugar elimination increases up to the second hour quite regularly after cane sugar, except in a few cases where the rate of excretion curves are similar after both glucose or cane sugar. More data on this point as well as on different types of glycosuria are being accumulated, but it already seems evident that sugar excretion rates run more consistently and regularly after cane sugar tests than do the sugar concentrations after glucose tests. In fact, the data so far at our disposal plainly indicate that rate of ex-

cretion gives more reliable information than does concentration concerning the sugar elimination of glycosurians and diabetics as well as normals. Incidentally these results also definitely justify our practice of employing cane sugar instead of glucose for carbohydrate tolerance tests.

Our data on other excretory substances than albumin and sugar likewise point to the clinical advantages of employing excretion rate instead of concentration as the criterion of elimination.

In conclusion, our thanks are due Dr. Fred Schattner and Mr. John Huizer for valuable assistance, and especially to Dr. F. M. Allen of Morristown, N. J., for his interest and kindness in providing clinical material.

SUMMARY.

Certain principles underlying the finding of albumin and sugar in urine are considered, and attention called to the fallacies of concentration and the advantages of excretion rate as the clinical criterion of elimination.

Easy methods for determining and applying the excretion rate are suggested and illustrated.

DR. PATTON—Diabetes or glycosuria of a harmless type is difficult for us to determine in many cases. The work of Drs. Folin and Blatherwick is too well known to need any comment from me. Dr. Folin has been with us a number of times and needs no introduction to you. Dr. Blatherwick has recently been brought into association with us by the Metropolitan Life Insurance Company. We can expect valuable suggestions from these two experienced teachers and workers.

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Folin-Blatherwick—Blood Sugar Curves 155

BLOOD SUGAR CURVES AFTER THE INGESTION OF 50 GRAMS OF GLUCOSE

OTTO FOLIN, PH. D. AND NORMAN R. BLATHERWICK, PH. D.
*From the Biochemical Laboratories of the Harvard Medical
School and the Metropolitan Life Insurance Company.*

Mr. Chairman and Gentlemen: Someone suggested that the last two pages of the proceedings here look like a time table, but those tables represent in tabular form the results of our investigation.

Some of you will recall that a year ago when we talked about blood sugar it was suggested that 50 grams of sugar could be used just as well as 100 grams and yield equally serviceable results. Dr. Blatherwick and I later discussed the subject and agreed that we should gather some data on that topic for a subsequent meeting, and these figures on the last two pages embody our results.

Now, the two things which are represented here and which I think you will understand better later when you get time to look over these figures are, first, that 50 grams of glucose gives perfectly typical blood sugar curves, and, secondly, that it is not necessary to take your subjects in a fasting condition. You see, as we grow older we try to become more practical and we have here, therefore, just two simple practical points, trying to make it easier and thereby also obtain more numerous blood sugar examinations than have as yet been obtained.

If you will look at these figures, you will find that the maximum blood sugar level is just about as high as one gets with 100 grams of glucose; also that that maximum comes very early, usually before or around 30 minutes after the giving of the sugar. Also it is to be learned, I think, from these figures something which we knew before, that the maximum height to which the blood sugar rises is not of any significance. It does not make any difference whether the blood sugar rises to 140 mg. or to 180 mg. The important point, and the only important point, is whether it will come down to the normal level at the

end of a certain definite period. In this case, we chose two hours as the period at which the blood sugar level ought to be back to the normal.

There is one difference between using 100 grams of glucose and 50 grams and that is that the more sugar you use the longer is it apt to take before you come down to the normal level. It was interesting to me—and I am sorry that I didn't notice it earlier in the work—that while in most of these 90 cases which we have examined, the blood sugar does come down to what you would ordinarily call the normal level at the end of the two hour period, there are, however, a certain number of cases in which the blood sugar has not come down to what I would call the ideal or the normal level for such an experiment. That is unfortunate from one point of view, but it is also rather satisfactory from another in that it proves that even 50 grams of glucose is abundant, since some of the individuals—and we have a fair number of cases of them—still had not come back to the original blood sugar value. The ideal condition which one should obtain in such a blood tolerance test is that the final blood sugar value should be the same, or rather lower than one obtained before the sugar had been given. As a matter of fact, most of our final blood sugar values are just about the same; many of them are lower than the original values, but some are higher. The highest figure which we have at the end of two hours after giving 50 grams of glucose is about 119 mg. per cent.

Since the figures do not quite come up to the ideal condition, it becomes necessary, of course, to lay down some practical figure that must be accepted as representing substantially normal conditions, and from the results obtained from these 90 cases, I should say that two hours after giving the 50 grams of glucose, it ought to be down to not over 120 or perhaps 125 mg. per cent. Of course, if we had continued these experiments for another half-hour, if we had taken two and one-half hours as our final period instead of two hours, then probably every one of them would have been down to the strictly normal level or below, but, after all, every half-hour counts if one wants to encourage

Folin-Blatherwick—Blood Sugar Curves 157

the making of the maximum number of experiments, and it does seem that one ought to be able to finish an examination of that sort within two hours. Instead of continuing the experiment longer, I think one could obtain the same result, although I haven't tried it, by using 40 grams of glucose. You see, it is a question of balancing the amount of sugar that is being absorbed with the time that it takes the organs in which to dispose of it.

Quite lately, in fact after these tables were sent in, I began to pay attention to this matter a little more, especially when your Chairman began to get after me about sending in the paper. When these letters came, I began to look at my figures. Then I didn't quite like them; I didn't like the fact that all of them had not come back quite as low as they should, and partly bearing in mind what Dr. Exton and I discussed publicly here last year, I thought we would try cane sugar. We have tried 50 grams of cane sugar instead of 50 grams of glucose, and so far as the blood is concerned, this cane sugar seems to give even sharper results than does the glucose. In every one of the 8 cases examined, the blood sugar came down to within 106 or 108 mg. per cent within the two-hour period, so that from the standpoint of sugar tolerance based upon blood sugar analysis alone, I am at the present time inclined very much to favor the cane sugar which you were talking about last year, but I realize that that should not be accepted except on the basis of further work. Cane sugar, however, is ever so much easier to take and one doesn't encounter the difficulty with a certain number of subjects as when glucose is taken. Some have nausea and feel like vomiting in the glucose test. Also the cane sugar is always pure, whereas the glucose isn't always as pure as we could wish. It was because of the troubles which we had with the glucose, students not liking to take it, not feeling very well afterwards, that we also tried to give the sugar at different temperatures. The common way is always to give the glucose sugar at ordinary room temperature, but we thought we would try it by heating it up to 50 or 60 degrees or as hot as the ordinary persons could take it, hoping that that would improve the results, but as

a matter of fact, it did the reverse. It made no difference in the form of the blood curve and the hot solution was harder to take than the cold, which we might have foreseen, as a matter of fact, and it was not absorbed any more quickly, so that there is nothing to recommend a hot solution as against a cold one.

I don't know how many of you have looked at these tables, but there is one other point that I want to bring out, and that is the fact that we analyzed the urine as well as the sugar in many of the experiments. Our procedure was to get the student into the laboratory and collect the urine for exactly one hour, then to give the sugar and to take the blood sample, or rather take the blood sample and give the sugar and then to collect another one hour's specimen of urine in order to see how the rise of the blood sugar level in the blood during that first hour, which represents the maximum rise, would affect the sugar excretion. As a matter of fact, in the vast majority of our cases, the urinary sugar in the second hour period is substantially identical with that of the first period, but out of our 60 odd cases, we had 4 or 5 in which the urinary sugar of the second hour is very much greater than in the first period, so much greater that, of course, it would give and did give positive Benedict's tests for sugar. I point that out partly in order to remind you of the fact that we used to test for diabetes by giving sugar, by giving more than 50 grams of glucose and by concluding that if the urine contains sugar it was a case of suspicion, beginning diabetes at least, and all these cases in which we have a quite pronounced excretion of sugar in the urine, 4 or 5 of them, the blood sugar curve is perfectly normal and therefore they have nothing to do with diabetes. Now that is exactly what you will run into in so far as you depend on the urine. Those cases are different and most of them are simply various degrees of renal glycosuria.

DR. PATTON—Dr. Blatherwick, have you anything to say now?

DR. BLATHERWICK—I don't know that I can say very much. Dr. Folin has covered all the material very fully, I think. There is just one point I might mention. If you have time to look

Folin-Blatherwick—Blood Sugar Curves 159

over the tables carefully, you will note that the blood sugar at the 90 minute period is often lower than at the two hour period. In my series, which is in Table 1, that occurs in about 40 per cent, of the cases, and Dr. Folin says that it is about 30 per cent. in his series, which is a pretty good agreement.

These sugars were done in both cases by Folin's ferricyanide method. There is a difference also in the type of subjects used. Dr. Folin's subjects were medical school students of a comparatively young age; my subjects were varied, from a young age to up in the sixties. My subjects were taken from our bio-chemical laboratory and comprised technicians, secretaries, and porters.

TABLE I.

Blood sugar after 50 grams of glucose preceded by a regular meal.

Subject	Age	Weight lbs.	Time after breakfast	Blood sugar—mg. per 100 cc.				
			hours	Initial	30 min.	60 min.	90 min.	120 min.
Male—N. R. B.	42	178	1.75	100	145	118	101	98
Female—S. S.	27	129	Fasting	98	156	131	98	106
Female—S. S.	27	129	2.5	102	140	140	116	101
Female—A. L. P.	30	133	1.75	98	167	112	73	80
Male—A. A.	68	156	4.0	94	154	109	122	88
Male—C. H.	29	144	2.75	98	133	120	105	94
Male—F. G.	29	127	2.25	98	145	149	143	114
Female—A. O'B.	35	175	2.5	99	183	140	91	91
Female—E. J.	26	134	2.0	91	135	118	105	99
Male—L. S.	27	146	2.25	102	140	106	103	99
Female—J. B.	19	103	2.25	94	130	116	101	108
Female—P. B.	29	118	2.0	100	143	114	94	81
Female—C. G.	27	103	2.75	89	142	111	104	122
Male—H. W. L.	30	135	2.0	100	165	159	115	94
Male—H. L.	25	156	2.0	97	138	114	84	100
Female—E. D'A.	19	127	2.25	106	151	144	89	91
Male—P. P.	28	133	2.5	99	148	119	79	101
Male—C. M.	56	185	3.5	108	182	148	79	91
Male—W. C. N.	44	150	1.5	115	163	123	116	105
Female—D. B.	29	155	1.5	102	161	107	71	87
Male—E. L.	29	127	2.5	101	157	154	133	89
Female—E. F.	30	125	2.0	105	147	108	114	99
Female—M. E.	29	102	1.5	104	141	146	114	84
Female—E. Mc.	25	110	3.0	98	154	119	119	99
Male—C. L.	24	142	2.25	98	150	122	105	88
Male—T. K.	21	145	1.5	123	139	107	113	94
Male—W. J.	34	167	2.5	129	187	141	104	112
Male—J. W. F.	35	169	2.25	92	168	131	80	88
Male—F. G.	29	127	2.0	110	151	141	101	112
Male—N. C.	42	158	Fasting	91	179	130	96	91
Female—A. B.	23	132	2.0	94	156	135	123	96

TABLE II.
Sugar in Blood and Urine after 50 gm. pure Glucose by Mouth, in 300 cc. H₂O.

Name	Age	Wt.	Remarks	Blood sugar—mg. per 100 cc.					Urine sugar—mg./hr.	
				Just before glucose	½ hr. after	1 hr. after	1½ hrs. after	2 hrs. after	Just before glucose	1 hr. after
J. S. M.	22	150	Breakfast 2½ hrs. before.	87.2	170.	156.	90.0	100.		
N. E. B.	26	150	Breakfast 3 hrs. before.	91.0	127.	140.	90.0	90.0	26.4	26.2
F. J. O. S.	22	160	Breakfast 2½ hrs. before.	90.0	105.	125.	93.0	95.0	26.4	24.0
M. B. A.	23	135	Breakfast 2½ hrs. before.	111.	162.	131.	101.	100.		
L. W. B.	22	175	Breakfast 2½ hrs. before.	103.	133.	108.	93.0	90.0	25.4	20.5
M. J. B.	27	160	Breakfast 2½ hrs. before.	100.	160.	178.	84.0	80.0	18.6	26.0
G. S. B.	23	135	Breakfast 3½ hrs. before.	93.0	146.	123.	94.0	93.0	23.7	21.5
W. F. H.	23	150	No breakfast.	88.0	129.	99.0	83.0	91.0	27.1	25.5
A. H. R.	41	167	Breakfast 3 hrs. before.	94.0	190.	135.	120.	105.	15.0	24.5
C. E. Y.	23	166	Breakfast 2½ hrs. before.	105.	180.	122.	105.	104.	37.5	54.0
J. L. B.	22	166	Breakfast 2½ hrs. before.	107.	131.	100.	104.	94.0	26.4	30.6
C. E. W.	25	182	Breakfast 3½ hrs. before.	105.	125.	118.	123.	95.0		
J. H. G.	21	133	Breakfast 3 hrs. before.	92.0	157.	137.	92.0	93.0	25.0	25.2
N. T. P.	24	153	Breakfast 2½ hrs. before.	101.	185.	119.	119.	93.0	24.5	24.9
C. A. W.	22	165	Breakfast 2½ hrs. before.	92.0	169.	105.	110.	93.0	24.5	45.5
F. H. R.	22	153	Breakfast 2½ hrs. before.	101.	147.	118.	104.	105.	20.0	20.5
P. S. W.	22	169	Breakfast 3 hrs. before.	112.	159.	136.	113.	101.	27.0	25.5

Folin-Blatherwick—Blood Sugar Curves 161

TABLE II, continued.
Sugar in Blood and Urine after 50 gm. pure Glucose by Mouth, in 300 cc. H₂O.

Name	Age	Wt.	Remarks	Blood sugar—mg. per 100 cc.					Urine sugar—mg/hr.	
				Just before glucose	½ hr. after	1 hr. after	1½ hrs. after	2 hrs. after	Just before glucose	1 hr. after
H. B. L.	21	142	Breakfast 2½ hrs. before.	88.0	154.	120.	81.0	88.0	35.0	30.0
A. A. M.	23	135	Breakfast 2½ hrs. before.	97.0	140.	111.	113.	97.0	31.0	
T. L. S.	24	132	Breakfast 2½ hrs. before.	94.0	162.	84.	93.0	94.0	32.2	38.5
A. A. Y.	22	132	Breakfast 2½ hrs. before.	93.0	160.	127.	92.0	93.0	26.7	71.0
J. V. C.	21	155	Breakfast 2½ hrs. before.	94.0	161.	127.	96.0	92.0	30.5	28.5
H. F. T.	22	145	Breakfast 3½ hrs. before.	93.0	177.	131.	106.	102.	32.5	275.
W. C. S.	21	135	Breakfast 2½ hrs. before.	94.0	143.	102.	110.	102.	28.3	40.0
F. D. O.	23	170	Breakfast 2½ hrs. before.	87.0	173.	83.	88.0	91.0	15.3	37.4
W. B. J.	25	145	Breakfast 2½ hrs. before.	89.0	127.	109.	92.0	91.0	46.0	44.0
J. J. L.	21	155	No breakfast.	83.0	106.	129.	123.	110.	24.4	22.6
R. J. S.	21	130	Breakfast 2½ hrs. before.	96.0	169.	96.0	88.0	91.0	37.5	35.5
A. P. B.	24	170	Breakfast 3 hrs. before. Sweating and nausea 1 hr. to 1½ hrs. after breakfast.	102.	153.	137.	96.0	92.0	26.0	31.0
F. J. H.	23	164	No breakfast.	95.0	127.	118.	111.	93.0	19.2	23.0
J. R. H.	22	175	Breakfast 3½ hrs. before.	94.0	161.	140.	112.	85.0	17.6	37.2
D. A. E.	23	160	Breakfast 1 hr. before.	88.0	121.	121.	105.	95.0	37.0	39.0
G. E. H.	24	165	No breakfast.	96.0	127.	145.	118.	86.0	17.3	19.4

TABLE II, continued.
Sugar in Blood and Urine after 50 gm. pure Glucose by Mouth, in 300 cc. H₂O.

Name	Age	Wt.	Remarks	Blood sugar—mg. per 100 cc.					Urine sugar—mg./hr.	
				Just before glucose	½ hr. after	1 hr. after	1¼ hrs. after	2 hrs. after	Just before glucose	1 hr. after
V. R.—Female	25	126	Breakfast 3 hrs. before.	94.0	157.	133.	117.	103.	29.5	26.0
L. P. C.	23	128	Breakfast 2½ hrs. before.	89.0	143.	101.	101.	90.0	25.1	22.0
A. R. C.	22	160	Breakfast 3 hrs. before.	105.	145.	111.	111.	94.0	23.3	23.0
B. W. C.	22	170	Breakfast 3 hrs. before.	105.	149.	104.	109.	103.	38.0	36.0
L. H. K.	23	160	Breakfast 3 hrs. before. No breakfast.	97.0	131.	127.	104.	111.	17.0	15.2
C. S. F.	21	175	No breakfast.	99.0	136.	133.	133.	119.	24.2	22.5
G. G. B.	23	135	No breakfast.	92.0	149.	143.	111.	92.0	17.0	16.0
R. S. W.	23	145	Breakfast 3 hrs. before.	101.	159.	117.	80.0	81.0	23.3	30.0
G. F. W.	21	192	Breakfast 2½ hrs. before.	90.0	137.	101.	85.0	86.0	29.0	28.5
G. D. H.	33	150	Oleated. Breakfast 3 hrs. before.	99.0	156.	108.	90.0	87.0	23.7	30.0
V. N.	40	160	Indian. Breakfast 2½ hrs. before.	97.5	133.	121.	105.	97.5	24.0	24.0
W. H. H.	22	143	No breakfast.	90.0	144.	123.	109.	89.0	19.0	20.2
G. H. McS.	22	167	Breakfast 2½ hrs. before.	81.0	113.	130.	90.5	90.0	26.7	22.8
S. M. D.	23	142	Breakfast 2½ hrs. before.	85.0	156.	100.	106.	83.0	23.3	20.3
J. W. C.	23	150	Breakfast 3½ hrs. before.	95.0	154.	112.	90.0	82.0	35.0	29.5
R. A. D.	25	183	Breakfast 2½ hrs. before.	111.	146.	116.	108.	111.	149.	120.
R. A. MacO.	27	150	Breakfast 3½ hrs. before.	93.0	156.	135.	93.0	84.0	20.3	56.7

TABLE III.
Comparative Glucose Tolerance Tests.

Folin-Blatherwick—Blood Sugar Curves 163

TABLE III.
Comparative Glucose Tolerance Tests.
50 g. pure glucose by mouth, in 300 cc. H₂O—With vs. without breakfast—Hot glucose sol'n
(50–60°C) vs. cold (i. e. room temp.)

Name	Age	Wt.	Remarks	Blood sugar—mg. per 100 cc.					Urine sugar—mg./hr.	
				Just before	½ hr. after	1 hr. after	1½ hrs. after	2 hrs. after	Just before	1 hr. after
O. W.—Female	27	155	No breakfast. Glucose sol. at rm. temp.	99.0	130.	96.0	100.	97.0	16.7	16.0
"	28	155	Breakfast 2½ hrs. before. Glucose at rm. temp.	89.0	121.	114.	89.0	88.0	29.0	29.4
"	28	155	Breakfast 2½ hrs. before. Glucose at rm. temp.	82.0	139.	121.	90.0	82.0		
"	28	155	Breakfast 3¼ hrs. before. Glucose sol. at 59°C.	91.0	160.	122.	87.0	79.0	25.0	25.3
"	29	155	Breakfast 3 hrs. before. Glucose sol. at 60°C.	86.0	160.	129.	89.0	83.0		
G. H.—Female	23	133	No breakfast. Glucose at rm. temp.	100.	172.	123.	104.	99.0		
"	23	138	Breakfast 2½ hrs. before. Glucose sol. at 19°C.	100.	160.	122.	92.0	91.0	24.5	28.1
"	23	138	Breakfast 2½ hrs. before. Glucose sol. at 59°C.	98.0	180.	119.	110.	85.0		
M. A. L.	31	145	No breakfast. Glucose at rm. temp.	94.0	167.	129.	97.0	88.0	35.4	34.3
"	31	145	Breakfast 3 hrs. before. Glucose sol. at 19°C.	98.0	176.	86.0	85.0	90.0	14.6	51.2
"	31	145	Feeling of nausea at 11:30. Breakfast 3 hrs. before. Glucose sol. at 60°C.	86.0	188.	121.	90.0	75.0	27.7	43.5
M. A. B.	25	165	No breakfast. Glucose sol. at rm. temp.	91.0	120.	94.0	100.	99.0	19.8	19.6
"	25	165	Breakfast 3 hrs. before. Glucose sol. at rm. temp.	96.0	123.	111.	104.	89.0	91.0	36.2
"	25	165	Breakfast 2½ hrs. before. Glucose sol. at 60°C.	100.	120.	109.	111.	104.	52.5	38.0
T. G.—Female	26	170	No breakfast. Glucose sol. at 59°C.	94.0	141.	135.	127.	87.0	16.8	19.0
"	26	170	Breakfast 2½ hrs. before. Glucose sol. at rm. temp.	84.0	138.	133.	116.	98.0	24.4	22.3
T. F.—Female	21	144	No breakfast. Glucose sol. at rm. temp.	100.	141.	114.	109.	96.0	14.9	17.0
"	21	144	No breakfast. Glucose sol. at 59°C. Had a very hard time getting it down.	90.0	129.	110.	108.	100.		

DR. PATTON—The Union Central has been one of our members that has always shown interest in improved methods. Their laboratory today gives us another illustration along this line. Dr. Muhlberg.

DR. MUHLBERG—Mr. President and Gentlemen: Assuming that very few of you have read the paper of Mrs. Behre and myself, I think perhaps it might be a good plan just to review rapidly what we have tried to do and what we have tried to demonstrate.

As a preliminary, we checked over the commonly accepted qualitative and quantitative tests for acetone and diacetic acid. The Ferric Chloride test detects only diacetic acid and the Nitroprusside test is more delicate to diacetic acid than it is to acetone and when you get a reaction, you can't be sure that you have a minute amount of diacetic acid and a large amount of acetone or whether the reverse is true. So we felt that it might be a good plan to review the various qualitative tests, and we found that really you can't depend upon any test for acetone in the urine unless you take into consideration diacetic acid. We decided to neglect hydroxybutyric acid because it is less significant and because there is no simple method to determine its presence.

Diacetic acid is changed pretty rapidly into acetone as the urine stands but the time seems to vary in different urines, being more rapid in some cases than in others. We found that in order to get any sort of qualitative test to give results in approximate values we had to use a distillation test, and Mrs. Behre worked out a very ingenious test. The distillate is made to pass over a piece of cotton having on it a drop of salicylic aldehyde and sodium hydrate. The diacetic acid is converted to acetone and will change the color of the bead to pink or red, which will indicate not only if there are acetone and diacetic acid present but will give a pretty good idea of the quantitative amount.

Then she tackled the problem of devising a quantitative test, which is done with color tubes. The quantitative estimates can be made in about eight or ten minutes and I think it is quite reliable.

Behre-Muhlberg—Acetone in the Urine 165

The next problem we undertook was to decide how much acetone occurred in normal urines. Hundreds of samples which we tested practically never showed more than about 1 mg. per 100 cc. of urine. That is an exceedingly small quantity, but occasionally there would be found somewhat more than that percentage. It must, of course, be remembered that acetone is very much like sugar in that it occurs normally in practically all urines in minute quantities. On the other hand, acetone may be increased by pathological conditions, such as nephritic conditions, vomiting in children and pregnancy; and it may be increased due to physiological conditions. If a person omits his luncheon or eats a light diet containing no carbohydrates the acetone will be increased.

What we are really after is the relation of acetone and diacetic acid to diabetes. Of course, in diabetes, acetone and diacetic acid are practically always found in the urine, unless it is kept in control by proper diet and insulin treatment. It is a fact that if a mild diabetic or certainly a moderate diabetic attempts to get rid of the sugar in the urine by cutting down the quantity of carbohydrates, he will increase the amount of acetone in his urine. The very mildest cases can, of course, if they are adroit enough in adjusting the diet, get rid of the sugar and also show no acetone. I have followed quite a few diabetics of the moderate type who were entirely sugar free but who showed quantities of acetone in the urine. We followed a few cases of diabetes under treatment with insulin and we found that the insulin controlled the sugar much better than the acetone. Most of those cases under insulin treatment showed on times quite a large quantity of acetone in the urine.

Now the situation is this, that by making tests for acetone, it may be possible occasionally to catch a person who is trying to get insurance by modifying his diet knowing that he is a mild diabetic. Dr. Joslin says there are one million diabetics in this country, perhaps two million. I wouldn't be a bit surprised to learn that some of these are finding it easy to procure insurance.

There is one question about the test that needs an answer and that is this: Is it worth while? The test isn't difficult to apply, those of you who have laboratories have technicians who can apply the test without difficulty. But actually you make hits only about once in a thousand times. Last year, for example, we made about 12,000 tests and we feel that in about a dozen cases we caught persons who, as far as we could judge, were mild diabetics or moderate diabetics who were trying to get insurance by modifying their diet. In some of those cases we had very strong confirmatory evidence. There is this peculiarity that five years ago we made a great many more hits than we are making now. It seems that information is passing through subterranean channels that the Union Central is not just the proper company to try that sort of a game on and we are getting fewer cases of that sort. Whether this test is worth while or not I don't know, but to make a hit once in a thousand examinations may be worth the effort since these cases are mostly big cases and are self-selected. The self-selected applicant usually doesn't limit himself to small amounts. We recently caught an applicant trying to get \$100,000 who unquestionably was a diabetic of moderate degree. We confirmed our opinion later on by further investigation and found that a month after he applied to us he was being treated in one of the hospitals in California for diabetes—yet the sample he submitted to us was sugar free and he denied any history of sugar in the urine, but he had, as I recall, 30 or 40 mgs. of acetone in his urine.

THE DETERMINATION OF ACETONE IN THE URINE
AND ITS SIGNIFICANCE IN LIFE INSURANCE
EXAMINATIONS.

JEANETTE ALLEN BEHRE, PH. D.
AND WILLIAM MUHLBERG, M. D.

*From the Biological Laboratory of
The Union Central Life Insurance Company.*

I. METHODS

In the past two years we have been directing some attention toward the subject of acetonuria, especially with a view to finding out how much significance the presence of acetone in the urine may have for life insurance work. We became aware of the limitations of the various tests for acetone and diacetic acid, and of the need for a simpler quantitative method than any of those which have been accepted as accurate, if the determination of these substances were to be made suitable for use in our laboratory. As a result, we have developed a qualitative test, which seems to have certain advantages over the usual tests, and also a quantitative method, designed to combine simplicity with enough accuracy to enable us to talk about acetone in numerical terms. We are now using both of these tests as part of our routine procedure, making the qualitative test on all incoming samples and the quantitative determination on those which show positive qualitative results.

Both methods give us information about only two of the "acetone bodies" which appear in the urine,—acetone itself and diacetic acid. We are forced to disregard the third acetone body—B—hydroxybutyric acid—in our routine work, because no simple enough method has yet been found for its determination. As this acid is rarely, if ever, found without certain amounts of the other two, it is probable that although our methods do not tell us the whole story, they do

give us a fairly definite picture of the degree of acetonuria present.

Before describing the methods, we might mention some of the disadvantages which we have found in connection with the usual tests.

The *Ferric Chloride* (Gerhardt's) test reacts only with relatively large amounts of diacetic acid and does not indicate the presence of the small amounts which may be very significant. Furthermore, it does not react with acetone itself at all, and since diacetic acid is gradually changed to acetone in the urine on standing, we will gain no idea from this test of the amount of the acid originally present. Ferric Chloride also reacts with any number of other substances, including many of the urinary preservatives.

None of the *Iodoform* tests are specific for acetone, and should be carried out only on specially treated and distilled urine.

The *Nitroprusside* test is very much better than these, but there are urinary substances which interfere with it also; and although it can be made very sensitive to diacetic acid, it is much less so to acetone. Since a varying amount of acetone is always found in connection with diacetic acid (the diacetic acid going over gradually to acetone as the urine stands), no quantitative estimate can be gained from this test.

Both of the methods which we are using are based on the reaction of acetone with salicylic aldehyde in strongly alkaline solution,—the reaction used in the old Frommer's test. Frommer's test was carried out on the urine directly, and has no real value in this form on account of the interference of sugar and other urinary substances. Nor does it show the presence of diacetic acid. We have found no reaction which gave satisfactory results when carried out in the urine directly. We have avoided this difficulty in our qualitative test by carrying out the reaction in the vapor above a test-tube, heated in a water-bath, and in the quantitative method by making a simple distillation which takes only a few min-

Behre-Muhlberg—Acetone in the Urine 169

utes. In both processes the diacetic acid is changed to acetone by the heating, and the results indicate the amount of acetone, both preformed and from diacetic acid.

The methods have both been described in detail in the *Journal of Laboratory and Clinical Medicine* (Vol. XIII; No. 8, p. 770 and No. 12, p. 1155, 1928) and we will be glad to supply reprints of the articles to any one who would be interested in reviewing them. We will describe the methods only briefly here.

In the qualitative test 3 cc. of urine are put into a test-tube and strongly acidified with 50 percent sulphuric acid. A small, thin square of cotton is cut and one drop of undissolved salicylic aldehyde (Eimer and Amend's Acid Salicylous, synthetic), followed by two drops of 32 percent sodium hydroxide solution, are dropped upon the center of it. The cotton is then inverted over the mouth of the test-tube with the spot formed by the reagents turned down toward the urine, and pushed down slightly into the test-tube to hold it in place. The tube is then placed in a boiling water-bath for eight minutes. At the end of this time, the spot formed by the reagents on the cotton will change in color, varying from a pink to a deep rose color, if anything over the normal amount of acetone and diacetic acid is present in the urine. Less than this will result in a yellow, or a yellow faintly tinged with pink, color. In this way we have made a sort of distillation of the acetone in the test-tube and collected the distillate on the cotton where it reacts with the reagents. A rough idea of the amount of acetone present may be gained from the depth of color on the cotton.

The quantitative method involves a regular distillation, but the whole process can be completed in about twelve minutes. Ten cc. of urine are put into the distilling flask, diluted with about 30 cc. of water, strongly acidified with sulphuric acid, and distilled into a graduated test-tube to a volume of 10 cc. Two cc. of the distillate are then pipetted into a tube of standard size, 2 cc. of 32 percent sodium hy-

droxide and two drops of Eimer and Amend's salicylic aldehyde (Acid Salicylous, synthetic) added, the tube shaken, and put into a boiling water-bath for three minutes. At the end of this time the tube is removed and the color compared immediately with a set of color standards in tubes of the same diameter. Directions for making these standards are included in the paper mentioned before. We would be glad to supply sets of standards to any one interested in trying the method.

The quantitative method as we have described it above will not determine with any accuracy, amounts of acetone below 1 mg. per 100 cc. of urine. In our experimental work we have found it possible to determine smaller amounts than this by starting with large volumes of urine (up to 100 cc.) and distilling these to 10 cc., thus concentrating the acetone. In this way it is possible to determine with more or less accuracy, the variations in the very small amounts of acetone found in normal urine.

There is one drawback to the use of both of these methods, in the fact that they do not give accurate results on urine which has been preserved with formaldehyde, or with hexamethylenamine, as formaldehyde decreases the color given by acetone in the reaction with salicylic aldehyde. Partly on this account, and partly because we were unable to have tablets made according to our formula, we have discontinued the use of the hexamethylenamine and salicylic acid preservative, which we were using until about a year ago. It must be said for this preservative that it was entirely satisfactory from every point of view, except the two facts mentioned above. It would be hard to find a better preservative in most respects. We are, however, now using tablets made us by the Mulford Company, each tablet containing 20 mg. Chinosol (oxyquinoline sulphonate) and 5 mg. of citric acid, with sodium chloride as filler. Our tablets seem to have only two drawbacks. If the amount of urine preserved with one of the tablets is very small, especially if it is alkaline, yellow

Behre-Muhlberg—Acetone in the Urine 171

crystals, due to the preservative, tend to form, and to interfere somewhat with the microscopic picture. And also the tablets are quite expensive. Otherwise, we have found them entirely satisfactory. It is interesting to note that they do not interfere with the fermentation of sugar by yeast, if enough yeast is added. However, yeast growths have almost never been found in our incoming samples with this preservative. The tablets do not interfere with the acetone methods which we are describing, nor with any of the tests in use.

II. THE OCCURRENCE OF ACETONE IN THE URINE

(1) *The Acetone of Normal Urine.*

Traces of the acetone bodies occur normally in the urine. The concentration of acetone (including that from diacetic acid) is usually well below 1 mg. per 100 cc. of urine.

Out of 3,757 incoming samples of which we kept a record, 95.5 per cent contained less than 1 mg. of acetone per 100 cc. of urine, while 55 samples (about 1.5 percent of the total) contained 1 mg., and 113 (or 3 percent of the total) contained over 1 mg. of acetone per 100 cc. of urine.

We have determined the acetone content of the urine of three apparently normal individuals, on normal diets, at various times and find it always to be below 1 mg. per 100 cc., and usually below 0.4 mg. per 100 cc. The excretion per hour is from 0.05 to 0.2 mg.

Such figures correspond with others reported in the literature on the subject. (Hubbard, J. Biol. Chem. 1925, LXIII, 391.)

In our routine analysis, we have decided to consider any amount of acetone below 1 mg. per 100 cc. as normal and to record any amount above this, regarding it as at least suspicious.

(2) *Acetonuria Due to Diet and Starvation.*

It would, however, be entirely erroneous to assume that acetone concentrations above this amount are unquestion-

ably pathological. There are a number of ways in which the output of acetone by a normal person may be increased. The acetone bodies come largely from the fats, either ingested fat or body fat. In normal metabolism the fats are broken down to carbon dioxide and water, with the production of heat and muscular energy, or else stored up as body fat. In certain abnormal conditions, however, the body is unable to complete the breaking down process, which is interrupted at the stage where diacetic acid is formed from the long fatty acid chains. Apparently the organism is unable to continue its normal action beyond this stage and as diacetic acid can not be used as such, it accumulates in the blood and overflows into the urine. B—hydroxybutyric acid and acetone are believed to be formed secondarily from the diacetic acid. In some way, not yet clearly understood, this failure of the body to complete the oxidation of fats, with the resulting acetonuria, is connected with a failure to oxidize carbohydrates. That "the fats burn in the flame of the carbohydrates" has become a physiological by-word, and it has been added that "if the carbohydrates fires do not burn briskly enough the fat is incompletely consumed; it smokes, as it were", and the smoke is represented by the acetone bodies. It has been established that it takes the oxidation of one molecule of glucose to oxidize about 1.5 molecules of diacetic acid, or about 1 molecule of fat.

Any condition, therefore, in which the oxidation of carbohydrates is reduced in proportion to the oxidation of fats, will result in the appearance of increased amounts of acetone. Such a condition may be brought about in a normal person by a diet low in carbohydrates or by starvation, when the body starts to burn its reserve fat for energy without adequate oxidation of carbohydrates. This is all doubtless very familiar ground. A great deal of material has been published on the subject.

Some experiments which we have been carrying out on normal subjects and which we expect to describe in detail elsewhere

indicate that slight increases in acetone output, above our normal limit, may be noted after shorter periods of fast, or of carbohydrate starvation, than has generally been supposed. In one normal individual we were able to note a slight rise in acetone output in a single day after the simple omission of lunch or after a very light diet, containing no carbohydrates, during the entire day. Although such a rise was not produced as easily in other individuals, it is obvious that we can not consider acetone above what we have set as the normal limit as necessarily pathological. From our point of view, however, the appearance of acetone in abnormal amounts may be significant, even if it is due to "dieting" or fasting, particularly by an applicant for insurance, who is desirous of making his urine sugar-free.

(3) *Diabetic Acetonuria.*

In diabetes the metabolism of fat is deranged, due to the inability of the organism to burn carbohydrates, and acetone bodies appear in abnormal amounts in the blood and urine, much as they do in carbohydrate starvation. By the use of insulin the diabetic is now able to burn carbohydrates and to control the glycosuria, so that in our work negative sugar findings are always subject to the possibility that insulin has been used. The question of the effect of insulin treatment on the output of acetone bodies is an interesting one. As one would expect, insulin undoubtedly reduces the excretion of acetone, as well as of sugar, in diabetic conditions, but apparently the reduction of the two is not necessarily parallel, and there is some evidence that the acetone may lag behind the sugar, and that the acetone, although lower after insulin injection than before, is still in many cases present in amounts which are above the normal, even where the urine is sugar free.

We are hoping to study this question in more detail shortly. We have so far only been able to follow the output of urinary acetone of two hospitalized diabetics who were receiving regular injections of insulin three times a day. In

both cases there was a continuous output of acetone slightly above the normal in spite of the continuous insulin treatment. In one of the cases the glycosuria was completely controlled by the insulin, but in the other the urine was never completely free from sugar.

Of course such cases are hardly comparable with the cases which we are likely to meet in insurance work, as they were both cases of well advanced diabetes, but they at least show that a person receiving insulin may still excrete amounts of acetone above the normal, even if the sugar is under control.

(4) *Other Acetonurias.*

There are, of course, other conditions in which the urinary acetone is increased. In certain nephritic conditions acetonuria appears. In rather a large number of our samples which show acetone, there is also albumin, or a history of albumin is found. In certain auto-intoxications and poisonings and in fevers, there is acetonuria, as also in grave anaemia. Chronic acidosis, both in children and in adults, is associated with acetonuria. In pregnancy, it is usual.

III. THE SIGNIFICANCE OF THE ACETONE DETERMINATION IN LIFE INSURANCE EXAMINATIONS.

The particular significance which the acetone determination may have for life insurance work is a question which seemed to us to justify investigation. In addition to the experiments already mentioned, and some others of a similar nature, we have made an effort to follow up the records of many of the applicants whose samples showed acetone in abnormal amounts.

We first made a study of samples sent us by policyholders in connection with conservation work. Out of 3,757 samples, 168 showed 1 mg. of acetone per 100 cc. or over. Of these only 35 had over 0.2% sugar. Of those which showed 0.2% or less, the records of 118 were examined. In 93 of these (31 of which had 1 mg. of acetone per 100 cc., and 62 more

Behre-Muhlberg—Acetone in the Urine 175

than this) nothing very significant was found, although 22 showed a record of some impairment other than sugar, 5 having at some time shown albumin or casts, 5 admitting Bright's disease, and 3 tuberculosis in the family, 10 being overweight and 6 underweight or losing weight. Thirteen of these cases were women, one of whom was pregnant. In the other 25 samples which were investigated, or 21% of them all, we found definitely significant information, for 16 showed a record of sugar at some previous time, while in 9 cases a definite diabetic history was recorded. Out of these 25 cases, 6 had been investigated because of the presence of 1 mg. of acetone per 100 cc. and 19 had shown more than this.

We also examined the records of 122 cases among the samples submitted by insurance applicants, which showed 1 mg. of acetone per 100 cc. or over. In 50 of these nothing significant could be found; 51, or about 42%, had records of other impairments or unfavorable family histories; while 21, or 17%, showed a previous record of glycosuria or diabetes, or a diabetic family history. Eighteen of these had shown over 1 mg. of acetone per 100 cc. in the samples which had caused us to investigate the records. Of especial interest were several cases where acetone above the normal, without sugar, was found in the first samples, and where sugar appeared in the samples submitted after the dextrose test had been given.

All of these were cases where nothing would have been suspected as far as the sugar determination was concerned. In view of the limited nature of the records which we have at our disposal, the fact that from 17 to 21% of these cases were found on examination to have a definite history of diabetes or glycosuria seems well worth consideration.

As a result of what we have seen so far, we have come to the conclusion that the acetone determination may be of real assistance in life insurance examinations. It is obvious that its value does not lie in replacing the sugar determina-

tion, but in supplementing it. When the sugar is high the acetone determination has little to add to our knowledge, although the presence of acetone in conjunction with moderate sugar is pretty clear indication that the sugar does not represent a mere alimentary glycosuria. Acetone does not necessarily occur in pathological glycosuria. A large percent of our samples which show sugar do not show acetone (evidently because in these cases enough carbohydrate is still being burned to insure the burning of the fat, if the diet is well regulated); and conversely, a majority of the samples which show acetone do not show sugar. It is in these cases, or those where borderline sugar is present in addition to acetone, that the particular value of the acetone determination lies. Such cases may be the result of normal eccentricities of diet, but they may also indicate conditions of latent glycosuria, where the output of sugar is controlled by diet or by insulin treatment. It is also quite probable that in the early stages of diabetes, or in cases where there is a tendency toward diabetes and where the appearance of sugar is intermittent, samples may be found showing acetone without sugar. This point needs more investigation.

These acetone samples without sugar may also represent non-diabetic pathological conditions. It seems rather significant that we find acetone so frequently associated with other impairments, especially with kidney conditions. We hope to obtain much more information with regard to these non-diabetic acetonurias. The relatively frequent occurrence of acetonuria in women, and the relation of acetone excretion to body weight are also interesting points, which need more investigation. We are planning to continue the study of these various points.

DR. PATTON—Those of us who know Dr. Anton R. Rose have no need to be informed as to his interest in his work nor as to his value in the development of insurance laboratory practices. Dr. Rose will give a brief comment on the paper he is presenting

Turbidity Micro Method for Blood Sugar 177

with others of the laboratory on "Turbidity Micro-Methods for Blood Sugar". Dr. Rose.

DR. ROSE—Mr. President and Gentlemen: Those of you who are interested in the examination of blood sugar will get the details of the Prudential method in the galley proof. The paper is exceedingly brief as the method is brief, and, thanks to Dr. Exton's clear style, it is very clearly presented and to the point.

The chemistry involved is exceedingly complex and I don't pretend to understand it myself, but complex as the chemistry may be, the details are exceedingly simple. Our Mr. Huizer in the Laboratory, in learning the method after two or three trials checked Dr. Schattner to within a deviation of 5 per cent on successive finger prick samples. The method has been in use about a year or a little over a year, and we have run simultaneously with the Prudential method Folin's method in which copper is reduced and Benedict's method of picric acid. The relations between the Prudential iron method and the copper method are much more consistent than the relations between the picric acid and the copper. Between the copper and the iron we have a relation expressed by the ratio of 1.05 with a maximum deviation of 0.45, whereas in the ratio between the copper and picric acid the deviation was twice that much and the ratio 1.25. Sixty-seven per cent, of the copper-iron ratios are within 10 per cent., whereas half the picric acid-copper ratios are not within 20 per cent.

The iron method used in the Prudential Laboratory has the advantages of both picric acid and the copper method, plus the additional advantage that it is simpler than either. Dr. Schattner will be very glad to demonstrate the method to any who wish to see the method in its details. The method is so simple that we think that it could very well be used in the field. If, however, it isn't deemed desirable for the examiner to use a chemical method, our laboratory experience in preserving the samples indicates that the sample may be deproteinized and dessicated and mailed to the Home Office. This work is so incomplete that we are not yet ready to publish our results.

TURBIDITY MICRO METHOD FOR BLOOD SUGAR.

A. R. ROSE, PH. D.; F. SCHATTNER, PH. D. AND
WM. G. EXTON, M. D.

The Prudential Insurance Company of America.

In the original (1) publication of this method the statement was made that it gave results which approximated Folin's copper better than Benedict's picric acid test. Thus, a typical sample of venous blood gave a sugar content of 154 mg. by the 1927 technic as compared with 170 mg. by Folin's copper and 194 mg. by Benedict's picric acid method. That subsequent technical simplifications have not changed this feature of the test is evident from the following recent comparisons:

Picric Ac.	Copper	Iron
133	121	107
267	244	237
135	111	93
129	102	84
265	220	246
112	100	104
212	183	192
100	91	91
150	139	91
173	94	90
151	91	90
154	114	100
151	110	108
148	127	126
113	103	108
106	105	78
114	88	88
120	102	115
119	98	89
166	137	117
129	102	92
108	79	84

Thus, if 100 mg. of glucose per 100 cc. blood be taken as the norm for picric acid, the copper method gives 80 mg.,

1. Rose, Anton R.: Preliminary Report on Turbidimetric Methods for Sugar in Blood and Urine, Proceedings of the American Society of Clinical Pathologists, Washington, D. C., May 14, 1927.

Turbidity Micro Method for Blood Sugar 179

and iron by turbidity 78 mg. per 100 cc. when the capillary blood is properly collected and measured.

In principle the new method depends upon precipitating the proteins in undiluted whole blood with hydroferricyanic acid. This method of deproteinization does not entrap the sugar or otherwise vitiate results, and like picric acid has the advantages of employing a deproteinizing agent which remains in the filtrate to function as the substance to be reduced by the sugar. Any ferrocyanide which may form prior to the sugar reduction from decomposition of the reagent or non-sugar reductions in the acid medium is precipitated with the proteins.

Such filtrates give lower values than Folin-Wu filtrates and we think this happens because complex acids, such as hydroferri and hydroferrocyanic, and pyrophosphoric, remove some non-glucose reducing substances in blood which pass into the filtrates of other deproteinizing methods.

When the filtrates are made alkaline and heated the ferri are reduced to ferrocyanic ions, and these are precipitated with silver in the presence of ammonia which holds the corresponding ferricyanide salt in solution. The fine ferrocyanide suspension is then measured in the Scopometer.

Reagents.

A—The Ferricyanide Reagent:

Complex cyanide solutions do not keep well. We, therefore, prepare the reagent fresh each day. This is easily done by keeping on hand powdered potassium ferricyanide and a stock solution of 0.27 grams ferric chloride dissolved in 600 cc. 0.1 N sulfuric acid made up to one liter with water. To avoid the tedium of weighing we measure the potassium ferricyanide with glass capsules having handles of twisted wire. For 100 cc. of the stock solution (0.67 grams ferricyanide) the capsule is made by sealing one end of a glass tube 23 mm. long with an inner diameter of 6 mm. For 10 cc. of the stock solution (0.067 grams ferricyanide) the capsule has

an inner diameter of 3 mm. and is 11 mm. long. Either capsule satisfactorily measures the ferricyanide and the reagent is quickly prepared by dissolving ferricyanide in the stock solution.

B—The Alkaline Reagent:

Dissolve 2.65 grams sodium carbonate and 2.50 grams sodium bicarbonate in 500 cc. water.

C—The Ammonia Reagent: i. e., a 10% solution.

D—The Silver Reagent:

Dissolve 31.0 grams silver sulfate in 500 cc. of 2.8% ammonium hydroxide solution.

Procedure.

Transfer 0.1 cc. blood from the pricked finger to a conical centrifuge tube containing 2 cc. of (A) ferricyanide reagent. Stir with a glass rod to mix thoroughly and allow 5 minutes for complete deproteinization and diffusion.

Centrifuge or filter and transfer 1 cc. of the clear filtrate to a 17 mm. test tube; then add 1 cc. of (B) alkaline reagent and heat the mixture in boiling water 10-15 minutes.

Cool under the tap, add 7 cc. of (C) ammonia reagent and precipitate the ferrocyanide formed on heating by slowly adding 1 cc. of (D) silver reagent along the sides of the tube. This and the next step of inverting the tube slowly several times should be carried out gently and as uniformly as possible.

Place the tube in the Scopometer, and take the glucose equivalent of the scale reading from the calibration graph or table.

Turbidity Micro Method for Blood Sugar 181

Scopometer Scale Reading	Milligrams Glucose per 100 cc.
18	30
24	40
30	50
36	60
42	70
48	80
54	90
60	100
66	110
72	120
78	130
84	140
90	150
96	160
102	170
108	180
114	190
120	200
129	225
138	250
147	275
156	300
165	325
174	350
183	375
192	400
201	425
210	450
219	475
228	500

In conclusion, it will not be amiss to suggest that the reliability and technical simplicity of the test make it practicable for field use.

DR. PATTON—Most of these laboratory methods we have had this afternoon have referred to glycosuria or the diabetic case, and I know no more important or difficult class of cases for us as Medical Directors. To me it is one of the most difficult classes we have, to decide whether or no we are dealing with a true diabetic subject or not.

The discussion of these laboratory papers will be led by Dr. Stanley R. Benedict who has been present at our sessions a number of times. Many of us have come to know him personally and thus have added to the respect for his knowledge and

abilities that we had obtained from reading what he has written from time to time. Dr. Benedict.

DR. BENEDICT—Gentlemen, the list of papers which I have been asked to discuss is rather heterogeneous and I shall therefore have to frequently change the subject matter of my remarks.

The first paper, the photo-electric scopometer is, as Dr. Exton suggested, a glimpse into the future where the electric eye, the photo-electric cell, supplants the human retina, the reading of the instrument depending on the change of flow in the electric current due to light sensitive cells and no longer being dependent on the chemical changes in the substance in the retina, which is broken down by light. I think Dr. Exton is definitely optimistic that he will have it fully perfected in a year; there are very serious difficulties, but we wish him luck.

Dr. Exton's second paper, the question of the hourly rate of excretion versus the haphazard sample has been, I think, repeatedly brought to your attention, and he has in that paper presented a simple and practical procedure for calculating the output of sugar or of albumin on the basis of the amount put out per fraction of the twenty-four hours. I question somewhat whether that is going to be worth while as a routine in the collection of urinary samples, because the difficulties will be marked, that is, every time you increase the technic of preliminary insurance examinations, you decrease the general success of the procedure, and from that standpoint I question whether in the low sugar values, the probable non-diabetic cases, that it is going to be warranted as a routine procedure. If such a procedure can be worked out without interfering with the applicant too much, it should be worth applying in sugar analyses.

I question the value of such a procedure applied to albumin. Theoretically I see no reason why it should be so applied. We know that the extent of the kidney lesion is rarely related to the amount of albumin excreted in the urine. We find cases of very advanced nephritis in which there is a mere faint trace of albumin in the urine. There are other cases, posture cases, where much albumin will be present in the urine of a man stand-

ing up and this albumin may disappear when the individual lies down. So I think that the evidence at present would hardly warrant going into the determination of albumin on the basis of hourly excretion.

The work of Doctors Folin and Blatherwick represents, I believe, a real step forward in the practice of routine sugar work in life insurance. It is an interesting thing to see that this is in a way a development of Frederick Allen's paradoxical law of carbohydrate metabolism which he put out, it must be, nearly fifteen years ago, and which, simply stated, is to the effect that there is no limit to carbohydrate assimilation in the normal organism except death. You can kill an animal with sugar, but you cannot make him eliminate an appreciable amount of it so long as he is non-diabetic. This is well known to be true. If you give a normal individual a hundred grams of sugar you may or may not detect a trace of sugar in the urine. If you give him 200 grams, you can probably detect a trace. If you give him 400 grams little if any more sugar is eliminated than when you give him 200; that is, there is no limit to the amount of sugar which can be utilized. And now Doctors Folin and Blatherwick have shown that the blood sugar story is similar to that of the urine sugar. The normal person takes care of 50 grams or 150 grams equally readily. If the sugar metabolism is inadequate, it can't rise to meet 50 grams any more than it can 100. You can show a failing sugar metabolism with the lower amount, and you can probably go down to 40 and possibly even 25 grams will give a marked differentiation. The differentiation between the normal and diseased man is not in the amount he can handle, but whether he can handle sugar in a normal way. I think Dr. Folin hardly emphasized the practical side of this as much as he might have. Many individuals cannot or will not take 100 grams of glucose, and cutting that in half is a very great item from the practical standpoint. Many can take the 50 grams with very little difficulty, and if, as it is indicated, cane sugar may be substituted, the gain will be even greater.

While as a general thing I think that new methods and new work should be very slowly adopted in insurance work, it seems to me that this is one of the cases where the general rule can be overlooked a little and that with perhaps two or three hundred more cases worked out you would do very well to switch over to the 50 gram basis, because the theory is all in favor of that and practice apparently confirms it, just as rapidly as the tests can be made.

The paper presented by Dr. Muhlberg on the determination of minute amounts of acetone impresses me far more from the theoretical side at the present time than from the insurance side. I think that the work carried out at the Union Central has very fundamental bearings. It seems to me to be a very important contribution. We have searched the world over for means to detect the pre-diabetic stage and here, in connection with the acetone, is at least one possibility opened up after failure. If, as they have found in a number of cases, the sugar falls below normal or to the normal level and you still have acetone detectable in abnormal amounts, it moves that very trying problem of early stages of the diabetic one step back, whether or not that routine is now adopted in insurance work. In view of the simplicity of the test, it might well be adopted. But laying that aside, I believe there is no question from the results they have so far reported that it should be adopted in urine examination where there is any reason whatever to question the carbohydrate burning ability of the individual. As Dr. Muhlberg pointed out, it is the usual thing to refer the burning of the fat to the carbohydrate molecule after the combustion of the fat has reached a certain stage, and assume that a certain ratio is necessary there. There must be a certain amount of sugar burned to burn a certain amount of fat. The technical methods developed in the Union Central laboratory will give us very simple direct accurate methods. I think the paper presents a very beautiful piece of work.

The paper of Dr. Rose brings up a point of some interest, I think, the question of the introduction of a still newer method

for sugar determination in life insurance work. In this connection, I would tend to urge a little caution, because in viewing the figures reported in that paper, I frankly am nonplussed at the discrepancies in some instances between the figures by the new method as compared with the older methods. Thus we find 139 mgs. by the Folin copper method and 91 by the new procedure. I should want to know why this is, before adopting the new method. That 139 is an abnormally high figure; 91 is strictly normal. Now why did the method give 91 against a well established method giving over 130.

We get to similar difficulties in the next sample, which gives picric acid 173, copper 94, iron 90. This method is tending to give very low figures. Why does it do this? The authors think it removes interfering substances. Before the method should be adopted, it would seem that that question should be answered definitely. This can be done readily enough by fermentation of the blood and trying the method and seeing if these interfering substances have all been removed. They are very nearly constant in value in the blood and I would therefore feel that that method is not, so far as the figures presented here are concerned, on an established basis but rather on an experimental basis. It has very definite advantages where sugar alone is to be determined in the simplicity of manipulation and I shall certainly look forward with interest to how it works out with a little more control.

DR. PATTON—We have had repeated instances of the interest taken by the Aetna in laboratory procedures, and Dr. Parker M. Cort of that Company will continue this discussion.

DR. CORT—Mr. President, the papers submitted this afternoon have borne in character a line of investigation in scientific thought that I hardly feel equipped to cope with. They all, however, express to us an inspiration on the part of the investigators who have contributed a great deal of thought and real value in their contributions toward a substitution perhaps of methods now existing and now employed—methods that might simplify and give us better practical results.

I would wish at this time to congratulate all the contributors upon their very excellent work.

There are certain practical phases in some of these papers that appeal to me and which I would like to bring up before you for discussion: First, is the matter of the new Turbidity Micro Method for Blood Sugar Determinations suggested by Dr. Exton. It has always in the past been my conception that when we started out to do blood sugar work for insurance purposes that we had assumed that the finger puncture was the ideal method of choice for collection undoubtedly because blood could be obtained with a minimum of inconvenience to the applicant and where small amounts are easily obtainable. Probably the first practical method of finger puncture and micro method of determination was that offered for our use by Miss Hunt of the Deaconness laboratory. This method depended upon a very careful laboratory procedure with considerable and frequent very accurate pipetting. We used this method in our laboratory on a great number of specimens for a year or over until Dr. Folin brought forth a very much more practical method of the blood sugar determination with small amounts of specimens and the use of a colorimeter for end readings. The result of this newer method was more accurate readings which were more easily obtained and were comparable in every way for all practical purposes to the more elaborate laboratory tests of Folin-Wu using venous blood. This method established itself as a practical method of procedure. This newer method, while dealing with small specimens, had eliminated the careful and frequent pipetting required in the original Micro method. The possibility of error had been reduced, and end results obtained that were more satisfactory, more quickly accomplished, and yet we had not increased the original size of our specimen.

Now I would offer as a thought in the discussion of this method suggested by Dr. Exton that it would appear as though we had reverted back to a type of procedure which involved the use of very small portions of a specimen at various times

through the process of its laboratory technique and very accurate and frequent pipettings were essential to an end result. It would seem that we have gone back to the laboratory technique of the original Micro method that had been considered obsolete after Dr. Folin gave us his more approved method. It sort of takes from beneath my feet the feeling that the method we employ of collecting blood in small samples, shipping them to the Home Office, and carrying them through by the Folin method, giving us end readings that are so materially higher than Dr. Exton has been able to obtain from his iron reaction is all wrong. I feel convinced that for all practical purposes that the collection of blood from the arterial net and submitted to the modified Folin-Wu determination, read by a colorimeter, works out better than I can hope with the method offered by Dr. Exton. I appreciate that presumably in offering this method he had in mind certain higher values that perhaps might be dependent upon other reducing substances and was prompted to devise some method which would give more accurate exact sugar content readings than we are getting by our present method. That is a laboratory procedure that I am not competent to discuss and could only be decided after a series of laboratory checks and controls had been made upon a known group of cases. As a general observation it would impress me that the variation between the readings obtained by his method and the readings that were formerly obtained cannot be entirely explained by the glycolysis that might reasonably occur in samples that have stood for any time before testing. It is obvious that if the picric acid and copper tests now employed are over-sensitive so that they reduce substances other than true glucose, then Dr. Exton's iron determination gives us more accurate glucose contents; but we must then readjust our present conception of normal values. I would prefer to see further results from a comparison of these tests before forming an opinion.

In justice one might conclude that Dr. Exton has offered not only through his new method and end result a thought that

might be checked by other laboratories. Confusion would follow and radical change in the figures now considered normal for blood sugar contents if one test should be substituted for another. Personally, I cannot foresee any great advantage through such a disturbance of figures we have all acquainted ourselves with and have fixed values in our minds. When a figure—for example: 120 mgs. of blood sugar—expresses the idea of normalcy, it is only relative. For all practical purposes it is of little importance if 10-20% of that figure is made up of other substances that are reduced by the same agent and confuse to a slight extent the exactness of the end readings. For laboratory accuracy it is highly desirable but for practical application when figures bear only relative values I would assume that its importance was over-emphasized.

The photo electric scopometer, with its photo electric eye, of course is a real scientific contribution. The application of the instrument I can foresee might be particularly useful in our laboratories to replace our present standard scale of turbidity indexes in our albumin tests. It might likewise be applied to the comparison of color necessary in reading the end results for our glucose test. The albumin turbidity test tubes are not particularly constant. They have to be replaced or renewed often. By the use of this instrument we would undoubtedly get more accurate readings and by this instrument more uniform results. Dr. Exton assures us that its ease of operation and rapidity of measuring solutions recommends it to replace the human eye. Measurements of solutions can be made in the original laboratory test tube in which the test is performed and this in itself is a distinct advantage. Its accuracy is superior to the eye because it is free from optical errors. The personal equation has been eliminated and turbidity or color can be mechanically gauged. It appeals to me as a wonderful and scientific contribution to our laboratory equipment for end results in the sugar and albumin tests are largely dependent upon the ability of a technician to match color or turbidity.

THE DETERMINATION OF ACETONE IN THE URINE AND ITS
SIGNIFICANCE IN LIFE INSURANCE EXAMINATIONS.

The paper of Dr. Muhlberg on acetonuria is particularly enticing to discuss. While I haven't sufficient faith in this test for acetone in the urine to feel that it could replace even in a small degree our present method of determining faulty sugar metabolism, yet we must admit that there is a certain co-relation between the presence of acetone or diacetic acid in the urine and a high blood sugar content. They go hand in hand. Acetone is the result of fat metabolism—the breaking down of the fats in our body. Sugar, on the contrary, is the breaking down or metabolism of carbohydrates. The familiar expression that one burns in the flame of the other very well illustrates how closely that are co-related. Certainly if we desire to get an accurate index of an individual's ability to handle his sugars, the test for diacetic acid or acetone would be a very valuable supplement.

Dr. Muhlberg points out that acetone bodies occur normally in the urine in small amounts. He has utilized the term "acetone" to cover diacetic acid and oxybutyric acid since acetone is the final state of these so-called bodies and any estimation of its presence would assume the others. The others are very rarely found alone and presumably any test for acetone would include the other two.

In excessive amounts of 1 mg. per 100 c.c. of urine or over it occurs often in those urines in which starvation exists. Clinically it appears often in severe diarrhoeas and its presence in excessive amounts is common in those diabetic conditions where the metabolism of the carbohydrates is greatly deranged.

Acetone bodies are dependent upon the normal process of fat metabolism. In certain abnormal states the body is unable to complete this metabolism and accumulations of diacetic acid or acetone appear, particularly in the urine. It is an interesting fact that an individual's inability to break up his fats in the body is in some way co-related to his inability to oxidize his carbohydrates. The relationship is subtle but one is necessary to the other. The fats burn in the flame of the carbohydrates

as Dr. Muhlberg well says, and this will illustrate the interdependence of one to the other. He points out that if one fire smoulders as the result of any impairment in the mechanics of our metabolism, there will result the appearance of excessive amounts of acetone in the urine. This gives us two interesting and valuable deductions—1. that an excess of diacetic acid or acetone may be an index of faulty carbohydrate metabolism; 2. that while you may reduce the sugar content in the urine and blood by a certain limitation of carbohydrate intake, the acetone appearing in the urine does not follow parallel with this curve. He further points out that by the use of insulin we may be able to completely convert the carbohydrates so that our blood sugar content will be normal; yet the acetone content in such states lags behind. Now, these two points alone recommend the test as a supplementary one for any complete sugar work. There are unquestionably certain large cases who have questionable sugar histories but who may not show sugar in the urine and whose blood sugar may be within normal limits by whatever method of determination you practice; but you have no assurance but what a certain amount of dietary control has existed for a day or two prior nor that the applicant has not taken insulin just prior to his blood sugar tolerance test. While a content of 1 mg. of acetone in the urine might not necessarily establish the diagnosis of faulty sugar metabolism that has been influenced by diet or insulin, yet it would be confirmatory of a suspicion that some starvation might have been practiced or insulin utilized. I feel that if this quantitative test for acetone were combined with our blood sugar work of today, it would add one more valuable method to our practice of underwriting these cases.

THE ADVANTAGES OF RATE OF EXCRETION OVER CONCENTRATION AS
THE CLINICAL CRITERION IN ALBUMINURIA AND GLYCOSURIA.

The paper by Dr. Exton on estimating the concentration of urines is a very valuable clinical contribution to our afternoon. The variables of time, volume, and concentration give us by this

method a rate of excretion that is important in estimating function not only for albumin elimination but for sugar.

It has been the practice of most clinicians and hospitals to measure and think in 24-hour amounts. From such a practice we have become adjusted in our conception of normal total 24-hour outputs. This opportunity of judging total out-puts is never given to the insurance companies. We are forced to base our opinion on one or more specimens obtained at random and often under the most unfavorable time conditions. We rely upon the element of chance and the number of specimens to represent for us the constant function of the individual. If one specimen should show a slight or large trace of albumin we may call for more and while it may be possible if these specimens are collected at certain times to be clear, yet we endeavor to judge the function by the number of specimens. This method is giving us no true index of real kidney condition. It is not dependent upon total or continuous elimination but upon casual specimens.

I am encouraged to feel that the method suggested by Dr. Exton of reading values from concentration might be applied by insurance companies with the modification that we adopt a longer time variable for the convenience of the Examiner and the applicant. The time variable of 3 hours would be preferable. Our Medical Departments would then have only to readjust our values for the estimation of their findings. We would then talk to each other in the terms of "one, two, or three" hourly concentration percentage rather than our present terms of "constant", or "intermittent" amounts. There is, I feel, a distinct clinical value that might be applied with good practical results to insurance work.

BLOOD SUGAR CURVES AFTER THE INGESTION OF FIFTY GRAMS
OF GLUCOSE.

It is particularly pleasing to have Dr. Folin's contribution following his warning of last year in which he said that he knew of no reason why we could not get satisfactory results from

50 grams of glucose as well as from 100 grams. He did caution us, however, at that time that we must not disregard the time element and if we plan to reduce the amount of our glucose tolerance test we must also cut down the time between the collection of samples following the ingestion of this test load. He has now furnished us with comparative figures proving his suggestions of a year ago and it is also bearing out his contention that high peaks mean little or nothing in glucose tolerance tests. I am not sure but what one would have to put a certain amount of limitation on these so-called high peaks. I believe he said 180 mgs. of blood sugar within one hour following a 50 gram glucose test meal might be disregarded. I feel that perhaps when amounts of 200 or 250 mgs. of blood sugar concentration are obtained in short intervals following the glucose test that perhaps we are obtaining a condition that would have to be recognized and could not be absolutely disregarded; but, after all, in doing sugar work the main thing to determine is the ability of the individual to carry whatever load you give him and to return to a normal point within a reasonable time and that is of the greatest importance in sugar work.

DR. PATTON—We had expected to have Dr. F. R. Brown of the New England Mutual give us their impressions of the laboratory questions raised by the papers we have heard. Dr. Brown has been called away upon business for his company and Dr. F. H. McCrudden of their Home Office will substitute for him.

DR. MCCRUDDEN—*The Photo-Electric Scopometer*—We have read with interest Dr. Exton's foreword on the Photo-Electric Scopometer. As he says, it offers a fascinating field for experimentation as to its future possibilities.

Granting the dependability and practical usefulness of the Junior Scopometer, it seems reasonable that this combination, with Photo-Electric cells, would be ideal.

We shall await with considerable interest Dr. Exton's further experimentation as to how practical it may become in solving some of our problems of medical selection.

Rate of Excretion—I feel that we are much indebted to Dr. Exton for his discussion of principles, his careful presentation of data, and interesting case reports.

There is no doubt that, if we know the rate of excretion, we have more information than we would have if we knew only the concentration. As to the value of the increased information in regard to albuminuria and glycosuria, I am inclined to be somewhat skeptical. Moreover, if this hourly rate of excretion is taken at random, will it not lead us into as bad a pitfall as will any other single criterion?

I feel that Dr. Exton has somewhat overdrawn the picture when he says that our present clinical practice is to be dependent on only one of the variables, time, volume and concentration, in order to emphasize the additional information to be obtained by considering all three. I do not think that concentration alone is ordinarily accepted as a sure guide in forming conclusions.

Is it true that, if the physician includes the effects of volume and time with concentration, that is to say, rate of excretion, he can compare his urinalysis reports strictly and truthfully if this rate of excretion does not extend over several hours' time?

I am inclined to feel that Dr. Exton has used an unhappy clinician in Mr. X's physician in order to emphasize what rate of excretion would have done if it had been used instead of concentration. I believe the former requires as much clinical judgment as does any other factor if it is to be properly interpreted and that it is not fair to assume that the physician who fails to properly evaluate concentration will necessarily be rightly guided by knowing the rate of excretion.

Moreover, I am doubtful as to the importance which is to be attached to the rate of excretion, either of albumin or of sugar. I am inclined to feel that the tabulation of the rate of excretion, in the case of protein, particularly, is of academic interest only. As to the rate of excretion of sugar, surely our blood examinations are of greater importance and guidance than the random determination of excretion rate. Is the case where there is a very low rate of excretion of protein any better than the case where there is a high rate of excretion? I do not think so.

Let me refer you to some work Dr. Dwight did, many years ago, on the urines of boxers. The urines of boxers were examined before and after bouts. These were divided into three classes: young men, who were at work in positions about town, who boxed at irregular intervals for the added income it afforded. The second class was a group of men who were locally engaged in bouts from time to time and who did no other work. The third group was the professional boxer who went about from city to city during the season. Let us consider first the young men who were engaged in clerical and other occupations. When they went into a bout the urine was universally normal. At the conclusion of the bout their urine showed a large quantity of albumin, it contained also blood and casts. The second group showed a normal urine before the bout and a little albumin afterwards. The professional showed a slight amount of albumin before the bout and very little increase after the bout. It would seem obvious that the boys who showed the large amount of albumin, blood, and casts after their fistic encounters were considerably better insurance risks than were those of either of the other two classes. Surely the rate of excretion would not be a guide in these cases, any more than would be the concentration.

Dr. Exton says that his data on excretory substances other than albumin and glucose point to the advantage of the employment of excretion rate, instead of concentration, as the criterion of elimination. This is in line with our views and action regarding creatinin. For a full discussion of this I would refer you to the article on Creatinin by Dr. McCrudden in "Life Insurance Medicine", Volume I. By this you will see that we are using the estimation of creatinin indirectly as an estimation of the rate of excretion of other substances.

Now if I may refer to our Company's attitude toward the concentration and rate of excretion as an insurance factor, I would remind you that indirectly we take both into account, since we do not overlook a possible high rate of excretion but low concentration, as we judge the former by the specific gravity and by determining the concentration of such factors as urea.

To recapitulate:

While I feel it necessary to doubt some of Dr. Exton's premises and conclusions, I believe much may be accomplished by further study of the excretion rate of substances other than albumin and glucose.

Turbidity Micro Method for Blood Sugar—Regarding this excellent contribution, I am unable to discuss the technical details involved. I would like, however, to ask one or two questions.

Dr. Exton states that the examiner can deproteinize and mail the filtrate to the Home Office. I would like to know if Dr. Exton has taken the filtrate of any of his cases and kept it for varying periods of time, under similar conditions as would prevail during the time the filtrate would be coming from the examiner's office to the Home Office, and has then completed the test and compared it with his original results. I would like to know this because of the rapid diminution in the sugar percentage when the blood sugar is examined by us by the Folin-Wu Method.

I would like to know if this method presents any difficulty in checking of duplicates. In asking this, I confess to a probable faulty technique, but we have had so much difficulty in checking our results by Folin's Micro Method that we have given up using the Micro Method entirely. Our results were so bizarre and our readings on duplicate specimens so varied that we were unable to put confidence in the method, hence, I would like to know if there is any difficulty from that line in following the test according to Dr. Exton's method.

Acetonuria—We have read with a great deal of interest Dr. Muhlberg's paper on "Acetonuria". He has given us a very complete and clear-cut resume of the various tests and their limitations. He has reminded us of our struggles with the elusive carbon atom and has recalled the mechanism of the production of the acid bodies from faulty fat metabolism.

When we first read Dr. Muhlberg's paper, we studied a thousand cases, taken at random and in sequence, in each of which a urinalysis had been made on account of some impairment. We divided these into two groups: One, those who had

a history of influenza, and, the other, a group in which the specific gravity of the urine was between 1021 and 1025, feeling that one might get similar percentages to those found by Dr. Muhlberg, if one took any chance criterion such as those mentioned. Our results, however, confirm the striking increases discussed by Dr. Muhlberg. Again, we checked over various impairments in a thousand cases, taken at random and in sequence, and, whereas these showed a history of kidney disturbance in 3.00% of the cases, Dr. Muhlberg found such a history in 10.76% of those which showed an increased amount of acetone. His figures for a previous record of glycosuria are 13.56% as against ours of 0.50%. These figures are very striking and certainly require careful consideration. We believe that the percentage of overweights which showed an increased excretion of acetone is also of considerable significance.

We feel that Dr. Muhlberg has shown us a sign-post whose direction is not yet entirely clear, but which we hope may soon point us further on our way to increased selection efficiency.

Since, in dealing with cases which have a sugar record, we make our decision on the strength of the blood sugar alone, irrespective of the presence or absence of sugar in the after specimen, we cannot see any present basis for acting upon an increased excretion of acetone. However, we hope to study a series of cases in which sugar tolerance tests are made with a view to ascertaining the effect of the test upon acetone elimination.

I believe the Association is to be congratulated upon the excellent and informative paper which Dr. Muhlberg has presented.

DR. MCCRUDDEN—Mr. President and Members: The other night over the radio I believe Einstein spoke at the fifty year anniversary of Mr. Edison's first work. Dr. Einstein, as you know, is the man who had so much to do with the invention or discovery or development of relativity. We have heard physics and chemistry come into life insurance. May I introduce relativity? The difficulty of measuring very large distances and

very high rates of speed appears to be that the measuring stick varies. Exactly here is the trouble. In measuring the amount of salt, sugar or anything that is passed in the urine, we have usually referred to the amount of water excreted as the measuring stick. We speak of the concentration, which merely means the amount of sugar, salt or anything excreted as compared with the amount of water. Water, your measuring stick, is varying all the time. You haven't a definite measure. But we have one fixed measuring stick. Dr. Folin found that the excretion of creatinin is constant from day to day and is also constant from hour to hour. If we use that as a measuring stick, we can eliminate one of the two variables, time and water excretion.

I think our group perhaps does not fully realize how much we owe to Dr. Knight of the Metropolitan Life Insurance Company and Dr. Folin for this kind of work. In the past, we life insurance Medical Directors, when we had to solve some problem in life insurance medicine, had to beg, borrow and steal from clinical medicine the work that we wanted. We had to go to the literature and take what we needed. We did not have the time and opportunity to directly go over the material we wanted when it was this kind of material that we needed, clinical laboratory material. Now, for the first time, Dr. Folin immediately takes up and does the kind of work we like done for us. Here is a very practical point which we would like to know. We use 100 grams of glucose. Why? In the literature in those thousands of such experiments 100 grams was used and willy nilly we had to use 100 grams. Dr. Folin takes 50 and I think quite probably that he will find that 25 will give us a suitable test.

In the second place, we had experiments only with glucose. We didn't have experiments with cane sugar. So Dr. Knight and Dr. Folin take the opportunity of making the matter simpler by using cane sugar.

In the third place, Dr. Folin, as you know, is working all the time on simpler and simpler tests of the blood sugar.

Now, those three points, using smaller amounts of glucose, using cane sugar and making simpler and simpler tests for blood sugar, are fundamental and are new departures in life insurance medicine. I don't think we all realize as much as we might the amount of work it takes to make 500 blood sugar analyses. It takes a long time and is a very tedious piece of work.

The question of the presence or absence of sugar in the urine after such a test Dr. Folin has touched on. It is a very important one. The results are very irregular, and so far as I can find out, there is no correlation whatsoever between the presence or absence of sugar in the urine and the blood sugar findings. We find plenty of sugar in the urine often in a case that we believe on the basis of blood sugar analysis is perfectly normal; on the other hand, sometimes in the case we think is diabetic or pre-diabetic we find no sugar in the urine.

DR. PATTON—Does anyone want to ask a question or add to this discussion? I find that some few of the members present have not registered in the book that was provided. Those who have not done so, kindly do that so that we can have a proper record. Our Secretary would like to have as accurate a list as possible.

The dinner tonight at the Robert Treat Hotel will start promptly at seven. We have decided to try a little innovation in not seating the members arbitrarily at definite tables. The tables will seat six persons. We are trying out this plan this year and so you are privileged to seat yourselves.

Dr. Exton, have you anything to add?

DR. EXTON—In connection with the paper on the excretion rate as a criterion, some of the speakers led me to believe that they gather that I recommended definitely the hourly excretion. Now, the paper gives no such meaning. The hourly excretion as a unit is one thing, but you can get that by taking the urine over two or three or four hours. The point is that you get the sample, measure the volume and divide it by the number of

hours, whether it is one, two, three, four, five, six or twenty-four. In fact, the longer the interval, the better is the criterion.

Now the hour is late and I will not attempt to answer in detail some of the questions that have been asked about the method. I will simply say that you, as practical life insurance selectors, know that when you get a specimen which contains a large amount of albumin, you don't want to hear anything more about the case; if it contains less, you are willing to get another specimen and consider it, but by using this criterion with all of its relativity you get a more accurate measure. It is not as relative as the concentration alone or such indirect ways as trying to get the same answer by estimating creatine, which we have tried and found not nearly so satisfactory as the rate of excretion.

The photo-electric scopometer paper needs no discussion, but I would like, not in the way of discussion but merely in amplification of Dr. Folin's paper, to say that we have used cane sugar for years with satisfaction and that our results are exactly in line with his results. In the galleys it doesn't appear, but in the paper we have included one graph of a series of carbohydrate tolerance test meals on the same individual after an interval of a week or so; in one test using cane sugar and in the other glucose. I would also like to state that no matter how carefully you get down the requirements or pin down the technic of the carbohydrate metabolism test, there are bound to be irregularities. As you know from the literature, many workers who aim at the greatest exactness recommend so many grams of glucose per kilo of body weight. Dr. Reginald Fitz of Boston in the *Journal of Metabolic Research* some years ago showed that the rapidity of absorption from the stomach of glucose solution was irregular and that no matter how carefully you performed the test, you were going to get irregularities due to variation in the rapidity of absorption. In fact, he worked out a method whereby he gave the people measured amounts of glucose per kilo body weight. He allowed the sugar solution to remain in the stomach a definite time; then pumped out of the stomach

the glucose solution that had not been absorbed and made his calculations accordingly. I mention that to amplify Dr. Folin's figures, because it seems patent that no matter how perfect a test may be or how well carried out, there must be irregularities due to differences in the rates at which the glucose solution is absorbed from the stomach. The previous diet, of course, also affects such tests.

DR. PATTON—That closes the discussion and our program for this afternoon. We want to start as promptly at 9:30 in the morning as we can, because while we have Dr. Dingman's paper slated for the first paper in the afternoon, we will probably reach him before we adjourn for luncheon.

(Adjourned).

SECOND DAY.

President Patton in the chair.

The Secretary announced that he had cast a ballot as instructed for the election of the officers and members of the Executive Council placed in nomination on the preceding day, as follows:

President—	Dr. William Muhlberg
First Vice-President—	Dr. Robert L. Rowley
Second Vice-President—	Dr. Charles L. Christiernin
Secretary—	Dr. Chester T. Brown
Treasurer—	Dr. Albert O. Jimenis
Editor of the Proceedings—	Dr. Robert A. Fraser

Members of the Executive Council:

Dr. George A. Van Wagenen
 Dr. Edwin W. Dwight
 Dr. Morton Snow
 Dr. Eugene F. Russell
 Dr. Ross Huston

These officers and members of the Executive Council were declared duly elected.

DR. PATTON—In arranging our program for this year, it was my feeling that as disorders of the digestive tract were so troublesome to us and entered into so many cases, if we could spend one-half day of our program in a discussion of those conditions, it would be worth while for all of us.

It has been my great good fortune to have had the opportunity of repeated conferences or, in the parlance of our profession, consultations with the author of the next paper. Dr. J. M. T. Finney is associated with the Prudential Medical Department in an advisory capacity and his counsel has been of much value to us. Added to his experience and skill as a surgeon, he has a rare ability to size up conditions. His opinions have been as

worth while for us as they have been for those who have consulted him as a surgeon. The diagnosis has been accurate and the treatment suggested has been curative. He is deeply interested in many things that are connected with the welfare of the country and finds time to take a prominent part in educational and religious affairs. It is, therefore, with great pleasure that I introduce Dr. J. M. T. Finney of Baltimore, who brings to us the paper on "Surgery of the Digestive Organs."

DR. FINNEY—Mr. President and Gentlemen: Needless to say, I appreciate the honor of being invited to address this Association. I am very glad indeed to see among its membership a number of my very old and warm friends, whom it is always a pleasure to meet and greet.

SURGERY OF THE DIGESTIVE ORGANS.

J. M. T. FINNEY, M. D.

From the life insurance standpoint, when considering the case of an applicant who gives a history of previous digestive disturbance of more or less severity, I take it that the questions of real importance to be considered are (1) the diagnosis; (2) the effect upon the life expectancy of the individual of the different digestive disorders, as shown by statistical tables based upon studies of large numbers of cases; (3) the effect, immediate and remote, of the various forms of treatment, both medical and surgical, upon the course of these different diseases.

Let us discuss these three general propositions briefly and seriatim.

Since the number and variety of digestive disorders, other than cancer, to which mankind is subject is so great, the brief time at our disposal will necessarily limit us to the consideration of a few of the more common and important varieties, such as gastric and duodenal ulcer, diseases of the biliary tract and appendicitis. Our discussion of the interrelation in diagnosis of these affections will be general, and largely the result of our own personal observation. In preparing this paper, it is not our purpose to present an exhaustive statistical study of the effect of digestive disturbance upon longevity. I would not presume to attempt such a thing before this body, composed, as it is, of men far more capable of doing such a thing than am I. I do want to emphasize, however, the difficulty met with in many cases of making a correct diagnosis as between the three conditions just mentioned, and the consequent uncertainty as to the proper course of treatment, and finally, to discuss the best methods of treatment of these disorders, as based upon a comparison of the ultimate results obtained by the more commonly accepted methods of treatment, as observed in a series of 510 cases.

Every physician or surgeon of any considerable experience knows how difficult, even impossible, it is at times to make a

positive diagnosis in a patient who consults him for indigestion, abdominal pain more or less vague and indefinite, associated with eating or not, as the case may be; varying abdominal tenderness, not always well localized, irregularity of the appetite and bowels, sallow complexion, possibly slight recurring jaundice, etc. This history may extend over a period of months or years; sometimes better, sometimes worse; the remissions never of very long duration, and the recurrences of gradually increasing intensity. This picture is a very familiar one, and one whose pathology is very difficult, at times, correctly to determine, for, in addition to the organic trouble present, sooner or later functional disorders resulting therefrom supervene, and a high grade of neurasthenia not infrequently results. Perhaps we can best illustrate just what we have in mind by giving a concrete example. A man of forty applies for life insurance. He gives a history very like that just related, strongly suggestive of chronic trouble, either in the upper or lower right quadrant, it is hard to tell which is more at fault. Sometimes the clinical picture as related by the patient himself, or as given by his medical advisor, suggests an ulcer, either in the stomach or duodenum, or biliary disease, possibly gall-stones, or even a chronic appendix. Some cases of hyperchlorhydria will closely simulate all these conditions. His trouble may have existed for a long time, in fact, he says he has not felt right for several years past. A year ago, his appendix was removed. For a short time, he seemed better, then all of his symptoms returned, even with renewed intensity, so much so that six months ago he was operated upon again. This time it was in the nature of an exploratory operation. The operating surgeon's report was that while no definite pathology was found in the upper abdominal region, nevertheless, in the hope of benefitting him, a gastro-enterostomy was performed and some adhesions in the region of the previous appendix operation were separated. Following this operation, he did not have the period of comparative relief experienced after the appendectomy, but rather his symptoms became more aggravated, especially the pain, nausea and vomiting, until now he leads a pretty miserable

existence, subsisting on a very limited diet, having frequent recourse to the stomach tube for relief; under-weight and under nourished; lacking energy of mind and body; self-centred; a confirmed neurasthenic. What about such an one as a life insurance risk? One sometimes is tempted to think that this type of individual would make an excellent risk, as he rarely dies, but goes on living indefinitely, leading a miserable existence himself, and making the life of everyone else with whom he comes into contact a burden.

Perhaps, in a somewhat similar case, instead of doing a gastro-enterostomy, the surgeon has removed the gall-bladder, with a result quite similar to that just related.

The questions of importance that emerge from this recital are (1) How really necessary (for life insurance or other purposes) is an absolute diagnosis in disorders of the gastro-intestinal tract? (2) What effect does the history, in general, of chronic digestive disturbances have upon one's status, as a life insurance risk? (3) How does the fact that an applicant has already been operated upon affect his rating? As has already been indicated, exclusive of cancer, the three affections that must always be borne in mind in any case of digestive disturbance are appendicitis, acute or chronic; peptic ulcer, gastric or duodenal; and biliary disease, with or without the presence of gall-stones. There are, of course, other disorders of the digestive tract that may occur, but they are relatively unimportant, and do not concern us in this discussion.

The medical examiner not infrequently has put up to him some very interesting, as well as difficult questions to decide, especially when to his other difficulties is added the necessity for a decision as to the effect upon life expectancy the particular trouble under consideration is likely to have. These questions cannot be decided satisfactorily by tables alone or by rule of thumb. Each case is a law unto itself, and should be judged on its own merits, after a careful study of the patient's history, the findings, operative and otherwise, and a thorough physical examination. This, of course, requires time and skill and training

upon the part of the examining physician. The routine office examination is all right, as far as it goes, and probably will suffice in the great majority of cases, but, in order to secure the best results for the policyholders, an up-to-date insurance company should be prepared to make the special examinations and tests necessary. This is imperative, and, I assume, in the best companies, such to be the case.

With regard to the differential diagnosis as between these three conditions, it is not always an easy matter to distinguish between them, even with the modern aids to diagnosis and the various special tests at one's disposal. But, after all, from a practical clinical standpoint, fortunately, it does not make so much difference as one might think, this inability to make a positive diagnosis. The question of vital importance, clinically, is "are the patient's symptoms sufficiently urgent to demand relief? Are they progressively increasing in intensity, or are they on the wane"? If the former, an immediate operation is imperatively indicated, and the diagnosis can then usually be established after the abdomen has been opened. If the latter, one can afford to wait, and the diagnosis may be made at one's leisure. All three of the conditions mentioned above may, sooner or later, become essentially surgical affections. It goes without saying, however, that in all of them, surgery should never be resorted to until a fair trial has been given medical measures, and it has been definitely determined that for this case, at least, they are without avail. Typical cases of these affections are easy of diagnosis, because each presents a fairly definite clinical picture. But the atypical case, and many of them are such, will require the exercise of all one's diagnostic acumen and, even then, in spite of all diagnostic aids, recourse must, at times, be had to the exploratory incision. To the uninitiated, it may seem foolish that there should be any very great difficulty in differentiating conditions affecting, as they do, anatomical structures so different and, relatively, so remote from each other as the stomach, duodenum, gall-bladder and appendix. Then, too, the pathological processes that are chiefly responsible for these clini-

cal manifestations differ widely, i.e., inflammation, calculus formation and the erosions (ulcers) so relatively frequent in stomach and duodenum. But, nevertheless, the pain so often referred, which is, perhaps, the most constant and characteristic feature of all three conditions, may be so misleading, both as to character, intensity and location, as to greatly confuse the differential diagnosis. This phenomenon is, of course, accounted for in large measure by the reflex action due to the anatomical arrangement of the nerves connected with the various segments of the spinal cord with which, thanks to the painstaking work of Ross, Head, McKenzie and others, we have come to be familiar.

It goes without saying, then, that the diagnosis is of prime importance in the consideration of any case for any purpose. It is the ability to differentiate between the various abnormal conditions that present themselves; to recognize the pathological changes that give rise to the accompanying symptoms; to appraise them at their true value and to apply the therapeutic measures indicated, that distinguishes the scientific physician or surgeon from the empiric or the quack. There can be no greater necessity for accurate, painstaking diagnosis in the profession than in the medical department of a life insurance company.

We want, in passing, then, to enter a strong plea for care and scientific accuracy in the matter of diagnosis. So much, in the matter of prognosis and treatment, depends upon this. It is not enough to say indigestion, dyspepsia, gastric neurosis, ulcer, etc., and let it go at that. What is demanded, in order to secure the best results for patient and company alike, is a diagnosis as nearly scientifically accurate as may be.

To be sure, every medical man of experience knows that, even with the exercise of the greatest care, it is not always possible to make a positive diagnosis. But in the case of abdominal affections, with which we are dealing, an exploratory operation is always possible. It should not be forgotten, however, that every unnecessary surgical operation is a reproach to surgery, and that no operative procedure, no matter how simple,

is entirely devoid of risk. Then, too, every exploratory operation may not surely furnish the information desired, as every surgeon knows to his sorrow, who has, with high hopes, opened the abdomen and, after careful search, has closed it, little wiser for his own or his patient's pains.

Some light may be thrown upon the question as to the effect of a surgical operation for any one of the three abdominal conditions before mentioned, by a study of the end results following each. Appendicitis may be quickly dismissed. If the vermiform appendix has been successfully removed, either early in an acute attack, before it has ruptured, or in the interval between attacks, which, in skilled hands, is done with only a negligible risk, no further trouble may be anticipated from it. But if drainage has been necessary, there is always the possibility of subsequent trouble from mechanical disturbances of the normal peristalsis due to adhesions, or to the development of ventral hernia through the drainage track, with all of its attendant evils. As a matter of fact, however, serious trouble, such as this, is fortunately, of relatively infrequent occurrence, but must always be reckoned with.

With regard to biliary affections, obstruction to the free passage of bile, the presence of stones or infection, acute or chronic, of the gall-bladder or ducts, constitute the chief surgical consideration. For some years, during the early development of surgery of the biliary tract, drainage of the gall-bladder with removal of the stones, if any were found present, was extensively employed. However, after a few year's experience, it came to be observed that recurrence of stone formation was frequent. As a result of this observation, cholecystectomy has gradually superseded cholecystostomy, with the result that recurrence after its performance has become so infrequent that a properly executed cholecystectomy, just as in appendectomy, may be accepted as a permanent cure. The same is largely true with regard to stones in the ducts, as thorough emptying of them is rarely followed by recurrence. Of course, in a small percentage of cases, stones are overlooked; or, where the gall-bladder has

simply been drained, they may recur; or the infection present at the time of operation may persist, and so on. In spite of all this, however, operation upon the biliary tract is followed, on the whole, by excellent results.

With the assistance of my Colleague, Dr. E. M. Hanrahan, a study of all the cases of gastric and duodenal ulcer,—five hundred and ten in number,—occurring over a twenty-five year period (1900-1925), in the Johns Hopkins and Union Memorial Hospitals, Baltimore, has recently been made. A brief summary of our findings follows.

The true evaluation of any therapeutic procedure is possible only when compared with the natural history of the disease against which it is used. In order, therefore, to determine the therapeutic value of a particular surgical procedure, such, for instance, as pyloroplasty or gastro-enterostomy, for use in gastric or duodenal ulcer, we may well use two standards of comparison,—(1) with the results obtained following medical treatment alone, to be accepted, owing to obvious uncertainties, with some reservation; (2) with the results following the use of other surgical procedures under similar conditions.

Inasmuch as the etiology and pathogenesis of peptic ulcer are not yet clear, all forms of treatment, both medical and surgical, are based upon accumulated experience and, therefore, largely empirical in character. Both medical and surgical procedures aim to relieve pain; to secure better drainage; to put the affected part at rest; to limit the amount of trauma by the passage of rough and irritating foods; to eliminate possible foci of infection elsewhere in the body. The patient who comes to his physician because of obstinate dyspepsia accompanied by periodic hunger pains, with their demoralizing effect upon his general morale, is of the class which chiefly interests us in this discussion. He does not present a surgical emergency. He is an individual who may or may not have a lesion of the duodenum which, if present and unoperated, may have to be nursed along for an indefinite period, possibly for the remainder of his life. The resulting disability may be slight, or it may be great, even to such an

extent as to incapacitate him for his ordinary routine of living. Whether or not surgery offers this patient more than medical treatment, is a question which interests doctor and patient alike, and whose answer we must find in the comparison of sufficiently large groups of cases which have been carefully studied and treated each way.

Unfortunately, for our purpose, many of the publications dealing with the medical treatment of ulcers make no differentiation between gastric and duodenal types. Most surgeons, however, feel that these two conditions present quite different problems and, therefore, should be dealt with differently.

Crohn, representing the modern medical point of view, states "In general it may be safe to say that conservative medical treatment permanently cures forty per cent, of its cases. During the course of treatment, one or two per cent. may die of hemorrhage, and probably even a smaller percentage of perforation." In considering the general prognosis of gastro-duodenal ulcer, he says "Death from hemorrhage, perforation or malignant degeneration occurs infrequently, and then usually within the first few months or years. If a case remains rebellious to medical treatment, and uncured after several years of symptoms, danger of death from the ulcer or its complications is remote. But, the chance of healing with permanent cure from medical treatment also becomes less favorable with the passing years". On the other hand, surgery, according to some of its most ardent advocates, will cure ninety percent. of the operated cases which survive operation, but the percentage of cases in the hands of the good average surgeon will run well below this figure, as low, perhaps, as eighty per cent. According to Coffey, the operative mortality rate in five of the best clinics in the United States varies from five and one-half per cent. to nine per cent.

Crohn's studies of immediate and late results of medically treated gastro-duodenal ulceration agree in general with those reported by other observers. He finds that the immediate results are very good, namely, eighty-six per cent, are, apparently, cured, only fourteen per cent, remaining unimproved. Most of the

recurrences take place during the second six months, when thirty-four per cent. of the apparently cured cases develop unfavorable symptoms. With succeeding years, new cases of recurrence are added, but no longer in the same proportion. Within four years, fifty per cent. of the apparently cured cases will have relapsed. From this very general comparison, we must be impressed by the fact that, after deducting the operative mortality, the percentage of patients who are immediately benefited, following either medical or surgical treatment, is essentially the same, but, as the length of time following treatment increases, recurrences appear more frequently in the medically treated group.

The choice of treatment may well be based on the age, temperament and economic condition of the patient, and the duration and severity of the symptoms. The patient who is over forty, who has had symptoms either constantly or even recurrently over a period of years, who, because of temperament or lack of means, is unable to follow the necessary dietary regimen, is more properly a candidate for surgery than the young man who has very recently developed signs of duodenal ulcer and whose occupation, means and temperament permit him to pursue a properly regulated and supervised course of medical treatment. If, after a fair trial of medical treatment, the disease has not been controlled, then surgery would seem to be indicated.

After consultation with our colleague, Dr. Thomas R. Brown, we have formulated the following as a list of valid indications for surgical intervention in gastric or duodenal ulcer, other things being equal. It would appear that this list of indications should satisfy both the physician and the surgeon.

Perforation, organic; obstruction; impaired motility; repeated hemorrhages; persistent pain or discomfort, due to local peritonitis, perigastric or periduodenal adhesions; unrelieved pyloric spasm; chronicity, repeated failure to bring about relief by medical and dietetic means, assuming, of course, that the treatment and after-treatment have been wisely advised and conscientiously carried out; economic factors, importance of limiting the period of disability in working people; the possibility of malignant degeneration of gastric ulcer, as suggested by a gradually falling

acid, persistent occult blood, slight lessening of appetite, strength, blood count, etc.; the possibility that the ulcer may be secondary to, or its symptoms kept up by disease of appendix or gall-bladder, or both. This triad being relatively common, and, as a rule, unamenable to the usual treatment of gastric or duodenal ulcer by medical or dietetic means, is often brilliantly cured by the removal of diseased appendix or gall-bladder.

Having reached the decision to operate, what type of operation offers the best chance of relief? There have been many procedures advocated from time to time. These, in general, include pyloroplasty, gastro-enterostomy, either alone or combined with excision of the ulcer, or with sympathectomy, and partial gastrectomy. Appendectomy or cholecystectomy are often combined with these. Excision of the ulcer alone may occasionally be employed. The benefit through pyloroplasty is believed to result largely from relief of obstruction and the abolition of pylorospasm, with the consequent reduction in the emptying time, together with the accompanying reduced gastric acidity. Gastro-enterostomy is also believed to bring about much the same conditions. The amount of irritation from food is diminished by means of the short-circuiting and it is claimed that reduction in gastric acidity may be favored through regurgitation of alkaline duodenal secretion and improved drainage.

Resection of large portions of the stomach wall has met with favor in many European and in a few American clinics. Its advocates believe that the resulting quantitative reduction in gastric acidity (which is due, not as originally stated, to the removal of the acid-bearing area, but to the removal of the stimulating factor, whatever that may be) diminishes the likelihood of subsequent gastro-jejunal ulceration. Partial gastrectomy is a formidable procedure, even in the hands of the expert surgeon. Before we can accept it in place of pyloroplasty or gastro-enterostomy, we should be convinced that the state of health induced in the survivors is appreciably better than that following the simpler operations. Of this, we are not as yet convinced, although it is true that in our small series of resections, the results have, so far, been excellent, but the number

is far too small from which to draw conclusions. In our hands the simpler operations have yielded almost as good results, consequently we prefer them.

By extensive division of the pyloric ring, as in pyloroplasty, an enlarged gastric outlet is formed, the ultimate diameter of which is limited only by the diameter of the duodenum. One great advantage of this reconstruction is the temporary abolition of the action of the pyloric sphincter. Frequently, there is to be observed an effect similar to that produced by dilatation of the anal sphincter for fissure in ano.

In 1927, we began a study of the late results following all operations which have been performed upon the stomach and duodenum in the Johns Hopkins and Union Memorial Hospitals, Baltimore, between the years 1900 and 1925. This study has been interesting in many ways. During that period, a great increase in all types of gastric surgery has occurred, but the most spectacular increase has been in the number of operations for duodenal ulcer. For example, between 1900 and 1915, fifty per cent. of all operations in our series were for gastric ulcer; forty-three per cent. for gastric cancer; seven per cent. for duodenal ulcer. Between 1920 and 1925 duodenal ulcer was the surgical diagnosis in forty per cent; gastric cancer in thirty-seven per cent; gastric ulcer in twenty-two per cent. We can offer no satisfactory explanation for this apparent increase in the incidence of duodenal ulcer. It is difficult, however, to believe that it is a real increase.

We believe that a period of at least two years should have elapsed before the late result of an operation may be determined with any degree of accuracy. Our study is based on the results noted in five hundred and ten cases,—two hundred and sixty operated on for gastric, and two hundred and fifty for duodenal ulcer. Of this number, sixty-one died following operation, a mortality rate of twelve per cent. One hundred and two, or twenty per cent., of the whole number were not traced. Subtracting these one hundred and sixty-three cases, (sixty-one dead and one hundred and two not traced), we have a new total of

three hundred and forty-seven cases of gastric and duodenal ulcer surgically treated. Of these three hundred and forty-seven cases, two hundred and ninety-eight, or 85.8%, were either entirely cured or definitely benefitted by surgical operation, while forty-nine cases, or 14.2%, were unimproved. In addition to these five hundred and ten cases of gastric and duodenal ulcer in which the diagnosis was actually verified by operation, there was quite a large group which had been diagnosed as "ulcer" in which the operation failed to verify the preliminary diagnosis, thus emphasizing the difficulty often encountered in making a diagnosis, to which earlier reference has been made.

Of the total five hundred and ten cases, seventy-one perforated,—forty gastric and thirty-one duodenal. Of these, sixteen, or 22.5% died, not all of which were operated upon, due to the unsatisfactory condition of the patient at the time of entering the hospital. Two facts of importance are brought out very prominently in this study, namely, that the age of the patient and the length of time the perforation has existed before operation profoundly affect the mortality rate. The younger the patient and the earlier the operation, the better. A study of the causes of death in those cases that have died six months or more following some operation for peptic ulcer, discloses the interesting fact that both cancer and tuberculosis occupy prominent places in the list of diseases named.

Among the more common causes assigned for early deaths following operation are obstruction, pulmonary complications, cardio-renal disease and infection. It must be remembered that these cases go back to 1900, when the direct result of faulty technique was much more in evidence than now. Attention should also be directed to the fact that this series of operations was done by both visiting and resident staffs of the two hospitals mentioned. It is fair to assume, then, that they represent a cross section of the average surgery of the country. Many, perhaps the greater part, of the cases were ward patients, both white and colored, just "the run of the mine", and, therefore, represent the most unfavorable class of clinical material upon

Finney—Surgery of Digestive Organs 215

which to operate. These facts may possibly help to explain the rather high mortality rate as compared with the figures from some other clinics. Be that as it may, our figures speak for themselves.

The operation most frequently employed was pyloroplasty. It was performed in sixty per cent. of all cases. It has been the operation of choice, when conditions have permitted its performance, for the simple reason that we believe that it least disturbs the normal physiological relations, while affording relief to the distressing symptoms. It should be stated that we make in every case a systematic survey of the abdomen and, at the same time, remove palpably diseased appendices or gall-bladders, a fairly frequent occurrence.

Of one hundred and fifty-eight patients in whom gastro-enterostomy alone was performed, twenty, or 12.6%, died. This seems an extraordinarily high mortality rate, but it may possibly be explained by the fact that we have included the first six months, rather than a short hospital stay, as the operative period. We have done this because mere recovery from the operation by no means necessarily constitutes a cure. Sufficient time must elapse before the real result can be satisfactorily determined.

Of two hundred and thirty-three patients in whom pyloroplasty was performed, fourteen, or six per cent., died during the first six months. The result in our series, then, would appear to indicate that the mortality during the first six months after gastro-enterostomy is twice as great as that after pyloroplasty. There were no immediate deaths following partial gastrectomy for duodenal ulcer in fifteen patients. We were unable to trace from fifteen to twenty per cent. of our cases following their discharge from the hospital.

Our figures indicate, therefore, as do the figures of most observers, that about ninety per cent. of those who survive the usual surgical procedures for peptic ulcer of stomach or duodenum are markedly benefitted. But, in all fairness, we must remember that this high figure is reached only after an operative or immediate mortality of about ten per cent. for the entire surgical group.

Again, if we start with two hundred patients and treat one hundred medically, and one hundred surgically, the experience represented by our figures would indicate that after sufficient interval,—say ten years,—sixty-three of the latter would be living improved, five would be living unimproved, fourteen would have died, ten soon after operation and four some time subsequent to it, and seventeen will not be traced. With fairly diligently pursued medical treatment, the one hundred cases not operated upon will doubtless show comparable results. There will be a higher percentage who are living unimproved, and we would not have had an operative mortality of ten per cent.

As regards the type of operation, pyloroplasty has proved in this series to be a safer procedure than gastro-enterostomy. If in one hundred surgically treated patients, pyloroplasty has been performed, only eight would have died at the time of the follow-up; five immediately after operation; three subsequent to it. This is a more favorable showing than that of the combined surgical group.

We would emphasize the fact that the choice between medical and surgical treatment for uncomplicated duodenal ulcer will depend very largely on the mental, economic and social status of the patient. Unless unusual circumstances affect the decision, surgery should be postponed until time has shown that medical treatment is of little avail. Our study of three hundred and eighty cases of duodenal ulcer treated surgically between 1900 and 1925 has shown that, while both gastro-enterostomy and pyloroplasty yield almost identical results in percentage of improvement among those who have survived the operation, the mortality, both immediate and subsequent, is lower after pyloroplasty, about two to one. Ninety-two per cent. of those patients who survived operation and were traced were well or improved for periods of from two to twenty years.

In this study, no account, of course, has been taken of the imponderable factors of physical and mental distress; loss of time from work, due to rest treatment, starvation and other medical measures which interfere with the patient's activities,

his ability to earn a living or his enjoyment of life. It will be generally admitted, I think, that these factors loom larger under medical than under surgical treatment. The loss of time and money resulting from long continued medical treatment will, probably, at least, balance the expense of a surgical operation. But, on the other hand, one must not lose sight of the mental distress, the post-operative pain and discomfort and the inevitable mortality attendant upon surgical operations. No conscientious surgeon will, for a moment, lose sight of these possibilities.

Hence, in the final analysis, what are to be the determining factors in deciding between medical and surgical treatment, or between one operative procedure and another? The conscientious physician and surgeon will carefully weigh all these factors in the light of accumulated experience and attendant circumstances, and decide accordingly. He will not be unduly influenced by custom or rule of thumb, or even weight of authority. He will decide each individual case on its merits, not by generalization or by standardization. Every case represents a separate individual problem, the satisfactory solution of which depends upon the exercise of surgical judgment of a high order in selecting the operative procedure best adapted to the individual case, which ability, in turn, can only be acquired by giving due heed to the lessons that are to be learned from the open-minded study of one's end results.

In comparing the medical and surgical treatment of duodenal ulcer, it must be borne in mind that, as a matter of fact, the two are hardly comparable at all, since surgery usually begins after medicine has failed. The favorable cases will respond to medical measures, while surgery, as a rule, deals with those cases in which medicine has proved unavailing.

Again, in this series, pyloroplasty, as the operation of choice, was done in the easier and more favorable cases, while gastroenterostomy was reserved for those cases in which, for some reason, pyloroplasty was not thought advisable, therefore, the more difficult. This may help to explain the favorable results under medical treatment, and the rather high mortality rate after

gastro-enterostomy in our particular series. In all fairness, therefore, in attempting to compute relative mortality rates for different methods of treatment of duodenal ulcer, due allowance should be made for possible errors.

To summarize,—we believe that a study of this series of cases of gastric and duodenal ulcer, representing, as it does, a cross section of the surgery done by the average surgeon, will warrant the following conclusions,—(1) the immediate results obtained by either medical or surgical treatment are essentially the same; (2) the late results favor surgical treatment; (3) pyloroplasty, in properly selected cases, is the operation of choice, by reason of the better end results following its employment.

Applying the results obtained by surgical treatment in the three diseases under consideration more specifically to the field of life insurance, the facts would seem to justify the following conclusions,—

(1) A well operated appendectomy would have no effect upon one's rating, except to improve it.

(2) The same is true of cholecystectomy, in uncomplicated cases of cholecystitis and cholelithiasis.

(3) In gastric and duodenal ulcer, operation in general offers a ninety per cent. chance of cure, with a ten per cent. mortality rate.

DR. PATTON—I am sure we are all going to profit from what Dr. Finney has brought to us from the direct clinical surgical viewpoint, and in order that we may have an additional clinical viewpoint, our next speaker is one of our best known clinicians in this part of the country. We are fortunate to have with us and to have a discussion of Dr. Finney's paper by Dr. Edward J. Ill of Newark, who has an established reputation as one of the leading surgeons of America. Those who have been privileged to know and consult Dr. Ill have always received good sound advice and have benefitted professionally as well as personally by their contact with him. The doctor for many years has been a member of the Board of Directors of The Prudential;

Discussion—Surgery of Digestive Organs 219

therefore he is interested in the membership and work of our Association and is not with us today simply as a clinical observer. Dr. Ill.

Dr. ILL—We have just heard from the lips of the master a study of the highest value. A study of an honest presentation of a difficult and perplexing problem. A study which will bear reading and rereading. We all recognize that he feels he is here for the patient and not the patient for him, and that every unnecessary operation is a reproach to surgery.

Dr. Finney once said, "That no conscientious surgeon can escape the realization of the great responsibility he is under, and contrary to common belief the responsibility continually weighs more heavily in consequence of a fuller appreciation of the true significance of a surgical operation."

In discussing the paper before us I am painfully aware of the responsibility that I am placed under. In his address to us he makes it plain that the internist should have a fair chance first to relieve or cure the sufferer or at least that he consult the internist. He wisely draws our attention to the difficulty in the obscurer cases. Only long continued studies of cases of ulcer of the stomach or duodenum will give us their life history and many of us believe we have not yet reached beneath the surface.

You gentlemen of the life insurance business have records to which we practitioners have given only slight attention and which would open the eyes of many of us to new facts or at least to new thoughts.

"There is too much loose talking during discussions," the late Dr. McMurrty once said to me. I trust I may not fall into this error.

Having had occasion some years ago to study the life history of fibroids I learned from our esteemed Dr. Hoffman that 28.7% of women over thirty-five years had fibroid tumors. After an exhaustive research I am able to say that these figures were the only trustworthy ones.

No one will deny the possibility of cancer developing in ulcer of the stomach, knowing that all chronic irritations are subject

to malignant degeneration. I speak of this because I have rarely seen it do so. My experience is that of many years. I did my first resection of the pylorus in 1885.

My experience received a serious shock, when McCarty's observations were published. Opinions in this regard differed greatly from Ewing's 2.2% and "he was not sure that some may have been the seat of cancer to start out with", to 68% as we learned from another source. In my mind that is the great question whether the ulcers are not cancers to start out with. The early deaths as I shall show later of those accepted for life insurance might be an argument in favor of this.

No insurance company could afford to accept an ulcer risk if ten or fifteen percent of all ulcers became cancerous. They would have discovered it before this. Also when they can take such risks, only long experience will dictate.

Dr. Finney once said that, "The material at hand may unconsciously be used to support a preconceived idea, which would detract immeasurably from a conclusion that is at best inferential." I confess to this but wish to state the conclusions were arrived at by a study of figures given me by your worthy president and not compiled by myself.

A perusal of the records of the Prudential Insurance Co., shows that 5163 policies were issued to individuals with gastric and duodenal cured(?) ulcers. Of these 3745 were called gastric. These records extend over eighteen years. Eighteen years should show something about the life history of ulcers.

Discussion—Surgery of Digestive Organs 221

TABLE I.
Ulcers: Duodenal (D) and Gastric (G).

UNOPERATED.

Insured in second year after cure.										
Policies	Deaths	Policy Years			Ages at Entry or Issue				Expected	% Actual
		1-2	3-5	6-10	15-29	30-39	40-49	50 and over		
(D) 120	1	1	-	-	-	-	-	1	.45	222
Cause= Tuberculosis, not lungs, in second policy year.										
Insured three to five years after cure.										
(D) 296	-	-	-	-	-	-	-	-	1.37	-
(G) 726	8	3	5	-	-	3	2	3	3.56	224
(D-G) 1022	8	3	5	-	-	3	2	3	4.93	162
Cause=2 Tuberculosis lungs; 2 appendicitis and 4 not stated. The 2 tuberculosis were aged 40-44 at entry and died in third policy year. The 2 appendicitis were aged 35-39 at entry and died in second policy year.										
Insured six to ten years after cure.										
(D) 182	1	1	-	-	-	-	-	1	.95	105
(G) 514	1	-	1	-	-	-	1	-	2.82	35
(D-G) 696	2	1	1	-	-	-	1	1	3.77	53
Insured two to ten years after cure.										
(D) 598	2	2	-	-	-	-	-	2	2.77	72
(G) 1240	9	3	6	-	-	3	3	3	6.38	141
(D-G) 1838	11	5	6	-	-	3	3	5	9.15	120

Looking over the statistics the first thing that strikes one is that the length of life is greater in the ulcer cases than expected in the normal individual. Please note I do not say life of the greatest pleasure. This is likely because of the careful living of the ulcer case.

Anyone who has ever attempted to analyze figures in medicine knows the difficulty he is up against. It is right here that we strike a snag. To discuss all phases of ulcer is an impossibility. Let me speak of but one or two.

There are more gastric than duodenal ulcers reported while experience has taught that the relation is about eight duodenal ulcers to one gastric. (Mayo-Alvarez). It is evident then that we must revise the figures and if there were 5163 of both then there will be 644 gastric ulcers. I am not juggling with these figures. I am trying to get at facts.

It appears that thirty-two died within five years and two died between six and ten years after the issue of the policy or thirty-four deaths in all.

The company does not issue any policy until the second year after a pronounced cure, neither in operated cases or unoperated cases. It also appears that there were no deaths in 120 duodenal cases operated on up to ten years.

TABLE II.
Ulcers: Duodenal (D) and Gastric (G).
OPERATED.

Insured in second year after operation.														
Policies	Deaths	Policy Years			Ages at Entry or Issue				Expected	% Actual				
		1-2	3-5	6-10	15-29	30-39	40-49	50 and over						
(D)	120	-	-	-	-	-	-	-	.55	-				
Insured three to five years after operation.														
(D)	381	4	2	2	-	1	1	2	-	2.44	164			
(G)	1153	9	4	4	1	3	3	1	2	5.62	160			
(D-G)	1534	13	6	6	1	4	4	3	2	8.06	161			
Cause=2 Tuberculosis of lungs; 1 cancer; 1 influenza; 9 not stated.														
Insured six to ten years after operation.														
(D)	319	2	-	2	-	-	1	1	-	1.56	128			
(G)	1352	8	2	5	1	-	3	2	3	7.41	108			
(D-G)	1671	10	2	7	1	-	4	3	3	8.97	111			
Cause=1 accident; 1 pneumonia; 3 organic heart; 1 tuberculosis of lungs; 4 not stated.														
Insured two to ten years after operation.														
(D)	820	6	2	4	-	1	2	3	-	4.55	132			
(G)	2505	17	6	9	2	3	6	3	5	13.03	130			
(D-G)	3325	23	8	13	2	4	8	6	5	17.58	130			

Out of 1534 duodenal and gastric operated cases there were six deaths in one or two years after acceptance; six deaths three and five years and one between six and ten years. Of the deaths reported two were of tuberculosis of the lungs, one cancer, one influenza and nine were not stated.

Out of 1671 insured for from six to ten years after operation we learn that two died in one or two years after acceptance. This entirely confirms what Doctor Finney tells us of his 510 cases. I am speaking of deaths sometime after operation. The causes of death given were, one accident, one pneumonia, three organic heart disease, one tuberculosis and four not stated.

Discussion—Surgery of Digestive Organs 223

In the unoperated cases we learn of one death in 120 policies after the first year of acceptance and that was tuberculosis. Again in 1022 cases eight died in three to five years after the acceptance of the risk. Three died in two years and five in three to five years after their acceptance, none died between six and ten years. There were two tuberculosis of the lungs, two appendicitis and four not stated.

The late deaths from operated cases were twenty-three and the unoperated ones were eleven. The unoperated ones had a chance to live twice as long as the operated ones. It is only right that we consider that the operated cases were likely those of a severer form of the disease and more subject to sequelae.

TABLE III.
ULCERS (Digestive)
Causes of Deaths.

	GASTRIC			DUODENAL			Grand Total
	Unoperated	Operated	Total	Unoperated	Operated	Total	
Accident (not war)...					1	1	1
Apoplexy							
Appendicitis	2		2				2
Cancer		1	1				1
Diabetes							
Heart, Org. Dis.		3	3				3
Influenza		1	1				1
Liver, Cirrhosis							
Nephritis (Bright's)...							
Pneumonia					1	1	1
Suicide							
Tbc., lungs	2	1	3		2	2	5
Tbc., other				1		1	1
Typhoid							
Warfare							
Other causes	5	11	16	1	2	3	19
	9	17	26	2	6	8	34

If we knew what the cause of death was of those whom it is reported as "not stated" the deductions might be easier. We only know that one had cancer but we do not know where. If we recapitulate we find there were thirty-four deaths in all of which fourteen died from a definite difficulty not referable to the stomach, which leaves twenty deaths from other causes. If we consider for argument, that these twenty died from cancer of the stomach, which is not at all likely, we learn that out of the 644 gastric cases about three per cent died of cancer. That is the highest possible figure one could make out. This figure is very much lower than we constantly read about. All these figures might have been severely changed if the records had gone to thirty years, instead as most of them did to only ten years. Or still better if we could know what eventually these 644 insured died of, we would learn a great deal more. Eventually we shall know.

I am speaking of this because I am well aware that life insurance will suffer if those who believe in a high cancer rate proceed as heretofore and do extensive resections. Already we hear of severe anemias of a similar type to the pernicious. The extreme surgery alone prevents a death rate of $5\frac{1}{2}$ to 9% (Coffey).

I have a letter before me in which the statistician of a large company reports that the medical directors rarely accept cases with the history of gastric or duodenal ulcer, and then only after from seven to ten years. The whole number accepted was 250 out of 414, operated and unoperated. This was out of 332,500 applicants.

From the statistics quoted there is hardly occasion for such severe penalty. Hospital records will be of no avail because such only show the severer forms of the disease.

The truth in insurance work comes through obtaining data of actual experience with a sufficient number of risks. We shall likely soon hear from them and their accumulated experience.

DR. PATTON—Dr. Cragin was devoting much of his attention to surgery before becoming a member of the Aetna Home Office staff, and we will now hear from him.

Discussion—Surgery of Digestive Organs 225

DR. CRAGIN—Mr. President and Members of the Association: The Association of Medical Directors is extremely fortunate in being able to get the benefit of Dr. Finney's vast clinical and surgical experience. Such discussions can only prove of greatest interest and value. We owe him and our President, for his foresight in getting Dr. Finney, our warmest appreciation.

It is a little difficult to discuss a clinical paper from a medico-actuarial standpoint, so many factors have to be taken into consideration. Dr. Finney has given us his figures on his own work so far as he was able to trace the cases. From a medico-actuarial standpoint it would be interesting to know what happened to the 102 cases which were not traced and also the unknown number mentioned in which the operation failed to verify the preliminary diagnosis. These would have to be analyzed in connection with the number of diagnosed cases on account of the possibility that some of these cases not suffering from ulcer and not operated might apply for insurance. However, Dr. Finney's treatment of statistics from the clinical standpoint is so eminently fair that they are particularly commendable.

Among the diagnoses not proven at operation might be included the acute erosions which simulate typical gastric ulcer but heal very rapidly under favorable conditions. X-ray may show these in the acute stage, only to find them healed at operation. We would have to handle these cases as ulcer in our underwriting.

Dr. Finney asks some very pertinent questions. How really necessary for life insurance examination is a diagnosis of the gastro-intestinal tract? Our routine examination fails to bring out in a great many cases a past history of gastric ulcer. After rather careful review of our ulcer cases we no longer take as conclusive the term "Indigestion" or "Slight stomach upset". We write back and obtain a careful history in all cases reported in this way. Frequently we are able to bring out a very suspicious ulcer history if not a conclusive one.

We realize that after all special tests are not always conclusive. A company that has been in the field for 78 years and has seen medical fashions wax and wane is probably a little skeptical as

to the value of any new method of diagnosis, and I think justly so, until Time has proven its efficiency. The insurance company cannot operate on an applicant for purposes of diagnosis. Therefore, the point of view of the operating surgeon and the medical underwriter must naturally diverge at this point. A perfectly justifiable prognosis on the part of the operating surgeon cannot be assumed by the medical underwriter. Surgical relief with a mortality of, say modestly, 12% is a questionable procedure from a sound underwriting standpoint. We cannot pick our surgeons. We have to take the general run throughout the United States and Canada and with the advent of the small town hospital and the small town operator who is anxious to rush in and do stomach surgery, we have a real problem on our hands. I have seen too many early pneumonias operated for appendicitis and credited to ether.

In all underwriting we have to have more or less of a set formula. Two plus two must always equal four. Therefore, our experience must be worked out on the diagnosis from the field of ulcer present or absent. We have been over a number of cases of deaths from gastric and duodenal ulcer in our company for the last 15 years. In reviewing the history of these cases as given on the examination blank there is absolutely nothing, with the exception of one or two cases of remote indigestion, that would lead us in any way whatsoever to suspect ulcer. One of our cases died within 12 days from the time the insurance was issued of perforated ulcer of the stomach. Another one died within a month from gastric hemorrhage. There could be some criticism on this last case in the underwriting for there was a history of remote indigestion but that was all. In the past five years these cases have been checked very carefully and we herewith append the table of our mortality on our sub-standard underwriting which is about the only answer that we can give to Dr. Finney's second question at this time:

Discussion—Surgery of Digestive Organs 227

Class	Years Exposed	Actual Deaths	Expected Deaths A.M. Select Table	Ratio	Expected Deaths S. S. Table	Ratio	Amounts Exposed	Actual Loss	Expected Loss A.M. Select Table	Ratio	Expected Loss S. S. Table	Ratio
Gastric ulcer without operation—												
A	4122	...	\$290,000	...	\$1,526	...	\$1,784	...
B	4523	...	159,000	...	940	...	1,319	...
C	6447	...	221,000	...	1,156	...	1,965	...
D	5224	...	319,000	...	1,464	...	3,235	...
Gastric ulcer with operation—												
A	2822	...	99,000	...	598	...	699	...
B	4234	...	212,000	...	1,104	...	1,549	...
C	5535	...	380,000	...	1,890	...	3,213	...
D	3512	...	155,000	...	653	...	1,443	...
Duodenal ulcer without operation—												
A	7756	...	920,000	...	7,108	...	8,309	...
B	5857	...	533,000	...	3,296	...	4,624	...
C	6035	...	431,000	...	2,201	...	3,742	...
D	3412	...	168,000	...	772	...	1,706	...
Duodenal ulcer with operation—												
A	5234	...	376,000	...	3,029	...	3,541	...
B	3	25,000	...	102	...	143	...
C	4835	...	356,000	...	1,702	...	2,893	...
696							\$4,744,000					

May I interpolate right here our method of underwriting. Gastric ulcer, operated or not, we do not touch until two years have elapsed; then we start in with the D rating, taking off a letter every year until at about the seventh year we take them for standard insurance. Duodenal ulcer, operated, we do not touch for one year. After the first year, depending on the operation and the operator, we rate either — to 180 per cent. or give them standard. The unoperated we do not touch for one year and then we start in with the high rating, 220 to 300 per cent. and gradually reduce it a letter a year until at the end of the fifth year we give them standard insurance.

As to how the operation affects the rating, we will have to rely entirely on the statistics furnished us by the follow-ups of the clinical surgeons.

Some 30 years ago Dr. W. L. Richardson, Professor of Obstetrics in the Harvard Medical School, was lecturing on instrumental deliveries. He remarked in substance: "Any obstetrician who has not invented a new forceps is the exception". He was one of the early specialists. We all had our laugh but on serious consideration one could always work out pretty practical ideas from his apparent jokes. Since that time there is no need to remark on the growing tendency of specializing; but to illustrate what he meant: forceps are long, short, bent at various angles and curves, with different traction attachments, etc. The same can be said of needle holders. In traveling about the various clinics and watching inventor and instrument and analyzing him carefully, observation shows it is pretty easy to size up the operator from the instrument. The instrument is not built for everybody to handle but is built for the operator. A large instrument usually means a large hand and vice versa. Traction attachments may mean puny musculature or elbows with a plus or minus carrying function. Applying these principles to surgical technique, the operator cannot be blamed for using a technique which in his hands produces the best results. One man can operate with success in a very limited space where another man needs plenty of elbow room. No single operation produces equal results in the hands of all operators. In underwriting we have to take the surgeon in Bangor, Maine, in San Diego, California, in British Vancouver, in Miami, Florida, and way stations and try and weld end results from various techniques and operations into a homogenous result.

We have taken a few groups reported by well known men and tried to work them into an analysis that would compare with Dr. Finney's. The results are as follows:

Discussion—Surgery of Digestive Organs 229

	<i>Original Series</i>	<i>Reported on</i>	<i>Cures</i>
Aetna Life Series (composite)	214	205	36%
	678	472	67%
	1000	1000	88%
	820	820	70%
	<hr/> 2712	<hr/> 2497	<hr/> 74% Average
Dr. Finney	510	347	85.8%
	<hr/> 3222	<hr/> 2844	<hr/> 75% Average all over country

Recurrences were not fully reported on but in the third largest group they were as follows:

At the end of	2 years	24%
	4 "	25%
	6 "	31%
	8 "	28%
	10 "	23%

These groups represent both medical and surgical treatment of gastric and duodenal ulcer.

One group of 205 cases reported 27% failures over a period of 5-15 years. Death 18%.

The end results were about equal in the medical and surgical treatment with the exception of those medically treated, which showed a slightly higher recurrence in gastric ulcer in the third largest group in the 8th and 10th years. The operative mortality varied from 7 to 12%. It is only fair to say that the overwhelming majority did not have Finney pyloroplasties.

Insurance underwriting deals with just one phase of medicine—a phase which is usually pretty largely neglected by the average practitioner—and that is prognosis. Prognosis is our art. Therefore, anything which pertains to making better prognoses is most valuable to this Association. We feel that we must aid and assist the clinician in every way possible to develop and set forth new methods pertaining to better prognoses provided they are proven and well tried. Having insured a person we have no hold on him other than to hope and pray that if he ever does get a gastric or duodenal ulcer he will fall into the hands of Dr. Finney. So long as the end results from either medical or surgical treatment are comparatively equal, it should make very little difference to us what the treatment is. Having surgical tendencies, I believe that surgery accomplishes better results

in ulcer cases than medicine and whereas the immediate results as shown in Dr. Finney's paper are about equal, it has been very pleasing to note from his figures that his surgery apparently has the better of the argument on the late end results; but we must confess that with the surgical treatment as given today in the United States, there are a very discomfoting number of recurrences. We agree with Dr. Finney that his operation apparently gives better results and it seems more logical than any other operation advised at this time.

DR. PATTON—The next member to discuss this paper was one of the leading surgeons of his city, but some years ago decided to discontinue that portion of his active work. Since then he has devoted most of his professional activities to life insurance and has been a valued member of his Home Office staff. I trust that he will favor this association with increasing interest and attendance, for I am sure his knowledge will be worth our while. I am very glad to have Dr. James P. Hutchinson of the Penn Mutual discuss Dr. Finney's paper.

DR. HUTCHINSON—Mr. President and Members of the Medical Director's Association: Dr. Finney has brought out in his paper many instructive points. A few of them I would like to comment on.

First; as to his 510 cases that he has discussed this morning, he has spoken of the "run of the mine" (meaning, of course, an unselected group as compared with cases of a highly specialized clinic), and has called our attention to his mortality of 12 per cent. I think any of us who have had surgical experience in the past, or are having it at present in the general surgical hospitals, would be very content if we, in pooling our results, could show an equally favorable mortality. As a matter of fact, he quotes Dr. Coffey "from 5 to 9 per cent", and I am wondering whether our rather optimistic point of view on surgical ulcer is not founded on a false premises and that we should rather take the higher mortality and from that draw our conclusions; for certainly it is a fair argument to say that if the

Discussion—Surgery of Digestive Organs 231

mortality is higher in a clinic, the end results probably will be less good.

I do not agree with Dr. Finney's feeling that the reason for the high mortality is due to the type of individual they operate on. I think he will have to make it the "run of the mine" of the surgeons that operate, and not the material that is offered to them, for I believe that the material that comes to a hospital has at least as good a chance of getting through a surgical operation in proper hands as the selected cases.

Dr. Finney has laid great stress on diagnosis and on that I am sure we will all agree; and on his diagnosis he founds, of course, the prognosis; and that is what we are chiefly interested in. Unfortunately, however, when a man goes to a clinic with the idea of finding out what is wrong, he gives the clinician a plethora of information and the surgeon has to simply separate the wheat from the chaff. When he comes to us, especially in these days of preliminary examinations, he forgets all his ailments and it is rarely that we get the proper information on which to found our prognosis.

In the brief time that I have had at my disposal before coming over here, I looked over our underaverage group, which can be of no scientific value to this body, because it is of short duration, the time of entry being 1925 and running to 1928, inclusive. We had too few cases to be of scientific value for two reasons: first, because we probably selected these cases with too great care; and second, in the ulcer cases we started with the theory that an ulcer case was never a standard risk. In these cases we scaled down our rating: in other words, put on a rating for all time, but reduced it to a 25 per cent rating after a period of years. The result was that these cases went off our books when the more liberally inclined Medical Directors decided that after two years, or ten years, a case is standard. With the exception of the ulcer cases, I fancy we treat this group as any other insurance company would treat it.

Now of these cases, it is interesting that in these four years, we did not have a death on our rated cases, but over that same

period and taking the entrants in general, we have 9 ulcer deaths, or 1.3% of the whole, and of these, only 2 gave any history whatever. One had had an appendectomy some 4 years before and one had had some vague gas pains 15 years before but claimed that since then he had been perfectly well.

We have had 12 gall bladder deaths in that same period, or 1.8% of the whole, and 10 of these cases were absolutely clean, two giving some vague history that might have aroused suspicion in the minds of some of you gentlemen, but which we overlooked.

We had 41 appendix deaths, or 3% of the total death claims, with at least 8 cases that gave some history of digestive disturbance.

The same is true of our disability claims, which from the period of their inception in 1914, to 1928, inclusive, give very much the same percentage. We had 18 claims of ulcer with no history; 16 claims of gall bladder, 1 giving a history of appendicitis; and 19 claims of appendicitis, with 1 giving a history.

We have had also a surprising increase in death claims on digestive cases. In a study of the causes of our increased mortality in the first six months of this year, all the increased mortality could be traceable practically to digestive disturbances. In other words, during the past six months, compared with the preceding six months, there was a 35 per cent increase in the mortality from digestive cases.

I agree entirely with Dr. Finney's contention that in cases of appendectomy and cholecystectomy after operation they are more favorable than before operation, provided the condition has not existed over so many years that they may have other complicating troubles.

I feel there are two important things about these cases, one of which Dr. Cragin has very well brought out. The first is that in our study of duodenal and gastric cases, we must choose our surgeon. There is no doubt whatever that in the past our appendix mortality was higher because of the lack of experience throughout the country with that form of surgery and the same condition undoubtedly exists today in the upper abdominal area,

Discussion—Surgery of Digestive Organs 233

especially in ulcer cases; so it seems to me that in evaluating any case we should first take into consideration the operator and have a very clear statement from him.

The other point that I think we should bear in mind is to make every effort to circularize our examiners on the importance of this question of indigestion. We are arousing among our examiners, I am sure, considerable irritation by our persistency in going back at them and asking whether this one attack of indigestion was the only attack; but we are continually receiving papers giving a history of an attack of indigestion, and after going back at the examiner, we find there is a previous history of it.

I am going to listen with great interest now to the medical aspect of these problems that we have been discussing, because I think the day the surgeon has intervened, we at least have a correct history and can arrive at a more or less definite conclusion of the value of these risks. I think the greater problem is what our rating should be on all these risks who are unoperated. We should know how that individual reacts to the idea of operation, because we all are losing cases of unoperated appendicitis which wait until the eleventh hour. The same is true in gall bladder disease and no doubt the same will be true in the ulcer cases. So I think the important part in this discussion is really what rating is justifiable on the unoperated cases.

DR. PATTON—Dr. Finney do you wish to say anything in conclusion?

DR. FINNEY—I don't know that I have anything to add to what has been said. I want to thank the gentlemen for their very kind and too complimentary discussion of my paper. I have simply reported what the figures show and will let them speak for themselves. I am going to report in detail a little later every death that has occurred, and again let each individual draw his own conclusions. It seems to me that that is the only way in which one can arrive at real facts. It isn't what one thinks that counts, it is what the facts show.

In comparing the statistics, it should be borne in mind that all the other groups included only the cases as they left the Hospital, while ours traced them for six months. In that six months, quite a number of deaths occurred, so that our statistics on the same basis would make a somewhat better showing. Without making any apologies at all, it is only fair to call attention to this fact. As you know, a good many cases are really not improved by operation at all. They live through it, they survive the operation and get out of the hospital, but they may die in a comparatively short time thereafter from some complication, pulmonary, for instance. That has happened in a number of our cases. We have included all these conscientiously, because we think, in order to tell about the real results of an operation, one ought to wait at least six months and see what is going to happen in that time.

Another thing was the rather high percentage of cancer and tuberculosis observed in persons operated for gastric conditions. Gastric trouble, of course, affects the nutrition very seriously, which may account possibly for the tuberculosis incidence, but I don't know just how it would affect the cancer problem, unless it does somehow predispose to it.

Then the immediate results in the older group of cases show a certain percentage of infections, which fortunately, we do not now have, and then, too, an undue percentage of cases of post-operative obstruction, due to the fact that a great many of them were drained, cases we do not now drain,—and they were heroically drained too, frequently great masses of gauze being stuffed in. All these factors come in, and they all have a certain effect.

We expect later to compare this series with the results of the last five years, and see what the difference is. It should be a very interesting comparison. One is shocked sometimes in looking over one's statistics, carefully gathered, to find how much at variance they are with the preconceived ideas that he has as to his end results. I have had that experience more than once.

Medical Aspects of Intestinal Diseases 235

The whole matter may be summed up in this way,—everyone recognizes that, so far as gastric and duodenal ulcer are concerned, recovery from the operation does not necessarily constitute cure. There is a very real difference. The war taught us that recovery from an operation or recovery from a wound might leave the patient a hopeless cripple. The question of restoration of function should engage the attention of the surgeon, as well as the recovery of his patient from the immediate effects of the operation. In other words, the acid test as to the benefit to the patient of a surgical operation is expressed in terms of the end results.

DR. PATTON—Following the surgical, we now desire to take up the medical consideration of this subject. This Association has depended a great deal upon the Canadian portion of its membership and we are again indebted to one of them. I know of no organization where that supposed line of separation is more of an imaginary one than it is in the Association of Life Insurance Medical Directors of America. Many of our Canadian brethren have continued their clinical work and thus have brought these opinions and experiences fresh into their life insurance medical responsibilities. We have in Dr. William F. Hamilton a valued member of our Association, one who has made a name and place for himself in private and hospital practice in Montreal, where he also is a member of the teaching corps of one of the leading medical schools in the world. Dr. Hamilton.

DR. HAMILTON—Mr. Chairman and Gentlemen: I shall take the privilege that the President has offered and make this as brief as possible. Before doing so, I wish to pay a tribute to Dr. Finney's remarkable paper this morning and to state how greatly I have enjoyed the frank presentation of his results.

MEDICAL ASPECTS OF CERTAIN GASTRO-
INTESTINAL TRACT DISEASES.

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In considering diseases of the gastro-intestinal tract—a topic of prime importance from the standpoint of life insurance risks—our president has laid upon me the task of dealing with medical conditions of the stomach, intestines, gall bladder and pancreas.

It is safe to assume that the members of this association since student days have been constantly and intently concerned with *Diagnosis*. Upon a correct diagnosis, prognosis and treatment intimately depend. The medical referee or the consultant for a Life Assurance company still finds the necessity for diagnosis, prognosis and treatment quite as insistent if not more so than in private practice and his reputation quite as much exposed to criticism. However from the standpoint of the Company's officer, with the diagnosis made and the risk classified, prognosis becomes the expectation of life of that class and the treatment of the case is the plan of assurance offered or the refusal to issue a policy.

As a practitioner and instructor in clinical medicine, I have long since held the view that diagnosis of conditions arising in other systems rested on a firmer basis than that made of diseases of the digestive tract. The evidence in digestive tract cases not infrequently lacks definition as compared with that made out in examining the circulatory or the respiratory system. The value of the anamnesis and physical examination and laboratory results is often much more difficult to assess, and for these reasons faulty conclusions result.

When one recalls the complaints of gastro-intestinal patients due to hypersensitiveness, incoordinated muscular contractions in various viscera and in different parts or sections of the same

Medical Aspects of Intestinal Diseases 237

viscus, the reactions to diet so little understood, the unsolved problem of intestinal toxæmia, of fermentation and putrefaction, the remote disturbed functions showing in eyes, skin, the respiratory system, the circulatory system, endocrines, etc. one is impressed with the infinite variety of complaints or symptoms and puzzled in separating the so-called functional from the organic cases and in giving to each group a proper value.

It is now about one hundred years since one of your countrymen observing the rough method in use for finding out the weight of loads of hay, etc. remarked that "an apparatus that can only get somewhere near the weight isn't good for much; if it is only pretty near right it is all wrong." So Thaddeus Fairbanks set to work and the platform scales were perfected and exact weights were determined. This association has been using comparatively rough methods for many years in assessing values of gastro-intestinal assurance cases. Our apparatus even now can only get somewhere near right, yet few of us will admit "it is all wrong". Our President in arranging the work of this session has doubtless been prompted by a desire to get a better expression of opinion concerning digestive tract impairments so that finally a system may be evolved and correct values determined.

DYSPEPSIA.

It is with some hesitation that one begins a topic with a definition yet for the sake of clearness it is often found necessary as in the present instance.

Dr. Adamson of Glasgow in his lecture on "Some Aspects of Symptomatology in Dyspepsia" defines the limitations of his title by saying that "Dyspepsia means difficult digestion in the stomach arising from morbid variations in its activities". Discussing the same topic Robert Hutchison considers it best to define dyspepsia as "discomfort of any kind arising during the process of digestion as the result either of organic disease in the stomach or of a primary disorder of its functions".

These definitions with others that might be quoted show that the tendency is to consider that organ whence the greater num-

ber of symptoms arises the seat of the disorder. That this is not the case is shown by the study of the pathology of the living as well as by an analysis of cases in large numbers. The statement that in that condition known as dyspepsia the stomach is rarely really at fault is a close approach to the truth. "The stomach", says Moynihan, "is an organ full of sympathy for other sufferers and it speaks so loudly that its voice only is heard".

Complaints which suggest dyspepsia are very common—epigastric discomfort sometimes amounting to pain, nausea, vomiting, diarrhoea, thirst, etc.

It is well to determine whether the trouble is in the stomach for there are several conditions with one or more of the symptoms mentioned above in which the stomach may be quite normal and the cause quite remote. One may recall an instance of the misleading sign of vomiting, where a normal appendix was removed in a case of cerebellar tumor later coming under my notice, the surgeon having been informed by the family physician of recurring attacks of right iliac fossa pain. Again the digestive seizures found in locomotor ataxia may masquerade for a time at least as dyspeptic attacks. Nephritis too may express itself by disturbances in the gastro-intestinal tract by nausea, dizziness, vomiting and diarrhoea. Thirst, nausea and diarrhoea occur among those symptoms incidental to exophthalmic goitre. Another condition familiar through clinical experience is that of angina pectoris. The complaints are of epigastric fulness, belching of gas, substernal pain worse after meals, and sometimes a statement that exercise tolerance is good. Reports of a few such cases unless carefully gone into are apt to give the impression of a gastric ulcer or gall bladder seizure, whereas a far more serious condition obtains in the circulatory system.

While reminding oneself of the possible remote sources of dyspeptic symptoms in the above references to the central nervous system, the excretory organs, the endocrine system and the circulation, one must not forget that conditions nearer at hand create similar disturbances—a pathologic gall bladder, a diseased

appendix or an heavily overloaded colon may bring out these gastric symptoms in a manner equally intense. A gastric ulcer may be quite confidently diagnosed on the history. This fact probably led Lord Moynihan to remark that "gastric ulcer was usually found in the right iliac fossa".

When one considers the stomach to be the seat or origin of the symptoms the next point to be decided is whether the case is a functional or an organic affection or disease.

Organic disease presents a picture more or less stable yet progressive. While "attacks" so-called may be experienced the patient does not enjoy complete relief as is so often the case in gall bladder or appendix dyspepsia. In cancer the symptoms may be very slow in making their appearance, and pain depending largely upon stasis or peritoneal involvement or both, may not be experienced even throughout the course of the disease. The physical signs, the x-ray and the chemistry of the stomach must all be appealed to for accurate diagnosis.

Reference to this point may be made when cancer of the stomach is discussed.

If we consider the life history of peptic ulcer we can say with Emery and Monroe it is "a truly chronic disease". (*Arch. of Int. Med.* 43, No. 6.)

When the practitioner confronted by a case of dyspepsia, is able to assure himself that it is not one due to such causes as are mentioned in our first paragraph,—reflex causes,—or to organic disease of the stomach itself, the inference is natural although not very exact that there must be a functional disturbance of this organ. At once the mists gather and the shadows deepen. Outlines fade and many clinicians hitherto comparatively clear in their thinking and speaking are lost in phraseology—and hypothesis. It is generally acknowledged, however, that the group of functional dyspepsias is shrinking perceptibly as our diagnostic methods improve.

Under the heading of functional dyspepsia it would appear that motor disturbances take the first place, and many sensory painful

sensations arising in the stomach find their best explanation in terms of hypertonus or hypotonus.

Hutchison points out that two types of disorder can be recognized.

1. An increase both of secretion and tone resulting from *vagal predominance*, the hypersthenic or irritable type of dyspepsia.
2. A diminution of tone often accompanied by lessened secretion resulting from *overaction of the sympathetic*—the so-called hyposthenic or atonic type of dyspepsia.

There seems to be a tendency to regard certain anatomical characteristics as indicative of these types. Hurst in a recent article calls attention to the presence of a "*gastric diathesis*",—the short stomach lying diagonally or almost horizontally, i.e. the hypertonic, hypersthenic, hyperchlorhydric stomach. Then there is the long stomach vertically placed having a normal or low acidity. This is found in those of the asthenic type with long chests and narrow intercostal angles—the hypotonic, hyposthenic and hypochlorhydric stomach. To conditions of the central nervous system many dyspepsia symptoms may be referred. Hence the term "nervous" dyspepsia which Adamson considers is more correctly described by the term "emotional" dyspepsia or psychasthenic dyspepsia. A run down, exhausted, neurasthenic state is readily reflected in the gastro-intestinal tract by anorexia, discomfort after food, nausea, constipation, and an atonic stomach and bowel. The motor and secretory functions are inhibited by the emotional or mental state, digestion and elimination are not carried on normally and a vicious circle is soon established.

For a long time gastropptosis or visceroptosis was considered as playing a large part in causing dyspepsia but now it has been shown that even the low lying stomach does its work provided the tone is maintained.

An analysis of 1,650 cases with gastric or dyspeptic symptoms, published by Drs. Blackford and Dwyer in 1924, shows that 47% were due to organic disease of the gastro-intestinal tract, 20% were associated with systemic diseases, 30% were set down

Medical Aspects of Intestinal Diseases 241

as belonging to the functional or nervous type, while 4% remained unclassified. Thus 67% of this fairly large group of cases are to be attributed to organic causes, a percentage sufficiently large to stimulate close medical investigation of all applicants giving a history of disturbances in the gastro-intestinal tract.

It would seem that the task of medical directors in this connection is to stimulate our examiners to get more accurate histories securing all available data, to keep ourselves fully informed as to the bearing of such data, separating the functional from the organic and, in the light of the experience of thousands of cases, make our decisions accordingly.

CANCER OF THE STOMACH; AND ITS RELATION TO ULCER OF THE STOMACH.

Cancer discovered anywhere in the gastro-intestinal tract or a history of such a discovery with or without operation constitutes an impairment at once prohibitive or practically so on any terms of assurance. Dingman in his book on Insurability has said that "few individuals who get cancer die of anything but cancer. Little wonder then that life insurance, if given, is given conservatively if not actually grudgingly". Speaking on the bearing of operative measures in cases of cancer this writer says: "whether clinical or insurance data of the future will prove that ten years freedom from recurrence may be accepted as demonstrating insurability is conjectural". On this point year by year clinical data increase and while certain aspects are favorable, yet when considered, by and large, carcinoma cases are quite unfavorable. In well selected cases of gastric cancer such as are discovered early before lymphatic invasion is manifest operative measures have been of great benefit. Fifty per cent of such cases reported by Eusterman from the Mayo Clinic were alive after three years. On the other hand operative mortality is high, and recurrences even after seven years have been reported.

We are concerned with the decision to be made in case of applicants of forty years and over in whose history there is found an account of digestive tract disturbance—dyspepsia. Difficult

enough in medical practice, yet in that of life insurance the decision becomes yet more difficult on account of the usual scantiness of the data. A search of the records for a clear history of cancer of the stomach in its early stages or a review of one's own cases which have been numerous, each leaves the same impression of the variability and vagueness of the reports. So much is this the case that it must be admitted, notwithstanding the advances of medical diagnosis, that there are no clinical symptoms or signs, there is no laboratory test, there are no x-ray results by which cancer of the stomach can be definitely diagnosed before serious anatomical changes have taken place. In regard to symptoms, quoting from conclusions after a review of several cases, Spriggs says: "The most important early symptoms are fulness, discomfort or pain not severe or continuous, but recurring persistently and arising in a middle aged or elderly person. The next symptoms are lack of appetite, dislike of food, or nausea with resulting loss of weight." Quite in contrast to this view is that expressed by Balfour in December, 1925, when speaking before the Western Surgical Association in session in Kansas. "Carcinoma of the stomach may exist without giving rise to any of the familiar symptoms such as loss of appetite, loss of weight and anæmia. *Most important of all diagnostic evidence is the absence of pain*". Dr. Balfour at the same time urges "that every gastric ulcer should be considered a precancerous condition".

As for signs, hæmorrhage is not significant of gastric carcinoma. Arising during cancer it implies *ulceration*, or advanced vascular changes. The stomach contents after a test meal may fail to show the characteristic lack of hydrochloric acid, or showing such, the cause may be that of anæmia or atrophy of the secretory glands. In like manner we may express our scepticism concerning the x-ray examination, held by some to be capable of giving a correct report in 95 per cent of cases. The earliest objective sign of cancer, that of local arrest of the peristaltic waves of the stomach, is found also in ulcer of the stomach as well as in fixation by adhesions. (We hesitate to ad-

Medical Aspects of Intestinal Diseases 243

wise our patients to submit to an exploratory operation on mere suspicion yet this sometimes seems justifiable.)

As has been already stated we are concerned with the decision to be made in cases over forty where suspicion is aroused. It would seem that in all instances where suspicion is clearly justified we must postpone awaiting developments or reject unreservedly.

It is now more than twenty-five years ago that the startling statement was made that upon 71 per cent of gastric ulcers, gastric carcinoma developed, a report made wholly upon histological evidence. This view was supported later by observations on more cases by the same group of pathologists. Needless to say these figures stimulated an increased activity of investigation of the subject in many laboratories but so far as may be discovered the close relationship expressed in this percentage has never been confirmed. Indeed upon histological evidence other pathologists supported by the combined opinion of a large number of representative gastro-enterologists, it would seem more in accord with the evidence to say that cancer and ulcer are related in from 5% to 15% of cases, and Clement R. Jones in 1923 claimed that "reliable statistics do not support the contention that cancer of the stomach is more frequent in patients who have benign ulcer than in those who have not."

A. Rendle Short in reviewing the discussion on this subject at the meeting of the British Medical Association in 1925 says that the general feeling was that a certain number of ulcers were cancerous from the start and a few ulcers go on to cancer, but that as a rule the two affections are distinct. Thus it would seem that in the light of evidence so far adduced the decision in ulcer cases need not be influenced by the chances of superimposed malignancy and the shortening thereby of the expectation of life.

ACHYLIA GASTRICA.

Since diagnostic methods are becoming so widely applied, reports of hitherto unrecognized or even unsuspected conditions are finding their way into our offices and are forming an im-

portant factor in deciding risks. In this connection the acid content of the stomach is reported not infrequently and cases of low acidity as well as a total absence of free hydrochloric acid come to one's notice from time to time.

"The gastric juice", says Adamson, "is a fickle product influenced by so many conditions." When this secretion is known to depart so widely from the normal as to be absent as in cases of achylia gastrica, the question as to the cause and the significance naturally arises. It is well known that the stomach secretion may fail fairly frequently in childhood when it is often regarded as due to a congenital defect. Intercurrent infections with associated gastritis or toxæmia may be accountable. In the fourth and fifth decades it is more frequently discovered. One may be reminded that a total absence of free hydrochloric acid may be quite symptomless and discovered only in a routine examination undertaken in neurasthenia, syphilis or anæmia, or again on account of Addison's anæmia or combined subacute sclerosis in another member of the family. On the other hand ¹⁵eructations of gas or painless diarrhœa—dyspeptic signs—or diarrhœa in the morning following meals, may find an explanation in the total absence of free hydrochloric acid.

When it is reported that an applicant for life assurance is the subject of achylia gastrica one's attitude toward such a case must be influenced by age, symptoms, state of the blood, and associated causative factors including family history, the history of previous gastritis or syphilis.

When this condition has been shown to persist symptomless for some years, other conditions being first class, the case might be accepted as standard or with a slight advance of age. Otherwise such cases should be postponed or rejected.

TUBERCULOSIS OF INTESTINE AND PERITONEUM.

Ten years ago, 1919, this topic was under consideration when Dr. Bartlett in his paper on "Insurability of the Tuberculous" introduced the discussion. Bearing upon the mode of dealing with such cases the following passages are taken from the dis-

Medical Aspects of Intestinal Diseases 245

cussion as reported in our Transactions and express the attitude of at least three Companies:

"We have never accepted cases with a history of tubercular peritonitis."

"I doubt if any one here would take a case of tubercular peritonitis following operation, certainly not until enough time has elapsed to prove that it was of no account."

"Cases with histories of tubercular abdominal conditions and those of tubercular peritonitis I would not touch."

As a primary disease tuberculosis of the bowel or of the peritoneum must be considered as rare, and thus in the majority of cases suspected the primary focus must be diligently sought. If the case is that of a female the pelvic history is of first importance. All favorite sites of primary tuberculosis infection are to be closely examined.

It is generally taught that the way or route of infection is the blood stream or in less acute cases the lymphatic route, the adjacent lymphatic glands, or an ulcerating focus in the bowel giving rise to local peritonitis over that area.

Not only on account of its serious nature but also on account of its incidence as a secondary focus, abdominal tuberculosis suspected or diagnosed constitutes a bar to life assurance. Occasionally, however, in the routine of the work of a medical referee cases with a history of abdominal disease turn up. These applicants have been operated on, a laparotomy done and on the best of authority tuberculosis of the peritoneum has been diagnosed—"the peritoneum studded with tubercles." After closing the surgical wound the patient in many instances makes an uneventful recovery. Six or eight years go by; the weight increases to average or somewhat above average. No lesion elsewhere is discovered nor has it been suspected by signs or otherwise. Doubtless a rare case has been cited but if the experience of members were taken perhaps such cases are not so rare as may appear to individuals.

It is only upon the evidence of a healed or quiescent lesion of long standing that any known case of tuberculosis is accepted. Apart from the reasons already mentioned, why consider tuberculosis of the peritoneum or for that matter tuberculosis of the bowel (enteritis) such a disqualifying condition as is indicated by the quotation above made? May not consideration and treatment be given these cases similar to that given to healed tuberculosis in any other system?

COLITIS.

A classification may serve to direct our discussion of this important digestive tract disease.

1. ACUTE CATARRHAL COLITIS.

often of brief duration
rarely recurrent
due to systemic infection or toxæmia;
of low fatality and rarely becoming chronic.

2. THE ULCERATIVE TYPE.

always severe
frequently recurrent and chronic.
due to cancer, dysentery and other causes, for the most part mixed bacterial infection.

3. THE DYSENTERIC TYPE.

This type has two sub-divisions

(a) amœbic—infection, the *entamoeba histolytica* of the tropics.

(b) bacillary—(1) Shiga bacilli, or
(2) Flexner bacilli, or
(3) Y. bacilli

found in tropical and subtropical countries.

4. THE MEMBRANOUS OR MUCO-MEMBRANOUS TYPE.

Chronic, usually afebrile
recurrent

arising primarily in some cases from protein sensitization and often associated with general nervous mental and nutritional changes.

Medical Aspects of Intestinal Diseases 247

A few moments may serve to pass the chief types a little more closely under review.

Disregarding the cases included in our first division we pass to consider the second type which as implied by its name includes a serious group of cases in which not only the general symptoms are pronounced but local destructive lesions are common. These are intestinal perforation, peritonitis and intestinal obstruction, complications requiring prompt and radical surgical measures. It is generally accepted that at bottom ulcerative colitis is an infective process but specificity has not been established. Bargen, Bassler and Hurst have contributed to the etiology of this affection. The first named claims specificity for a gram positive diplococcus as this organism has been discovered in pure culture from the bases of chronic ulcers and occasionally from early lesions elsewhere in the body. This organism injected into the veins of rabbits and dogs produces changes in the colon like those in human beings suffering from the disease.

In the third or dysenteric group, specificity has been fairly widely determined by examining the stools and the lesions. The amœbic cases are confined mainly to those living in the tropics while the bacillary types are more wide spread and occur in tropical as well as subtropical countries.

Mucous or muco-membranous colitis has always been conjectural from the etiological viewpoint. The treatment has been for the most part empirical. Recently, however, Vaughan, Duke, Andreusen, Hollander, not to mention the names of others, have contributed suggestive papers regarding the cause, and treatment based upon protein sensitization tests has been followed by good results in several instances.

Granting that allergy initiates the condition other factors doubtless account for the characteristics of disease seen in its later stages. Witness the changes subsequent to recurring attacks of spasmodic asthma, the original seizures of which are initiated by protein sensitization. Here we observe not infrequently bronchitis, emphysema, bronchiectasis, etc., as well as a general condition impairing the risk if not rendering it quite unacceptable on

any plan. Dingman says that this form of colitis may be handled as neurasthenia. May it not be equally if not even more to the point to suggest that this type of colitis may be handled as asthma cases, each in the early stages of the disease.

From the above it is clear that colitis presents variety in its clinical types. Needless to say the activity of any type precludes the favorable consideration of an application the subject of even the mildest manifestation. Usually, if not at all times, we have to deal with cases where an honest history or otherwise is recorded or suspected. The severity, the duration as well as the type of colitis are considerations of first importance. It must be remembered too that serious complications and sequelæ may arise in practically all types. The well recognized tendency to recurrence and the latency of complications alike suggest the necessity of a careful medical examination and rigid selection. Cases of ulcerative colitis and amœbic dysentery would seem to head the list of serious impairments requiring the greatest care and the longest period of freedom from signs and symptoms before accepting. In the less severe cases two or three years of health with return to normal average weight should in most instances allow of life insurance at standard rates. Possibly, too, disability coverage may be granted under these conditions. In those cases with complications requiring operative measures a much longer time may be necessary before they can be accepted even as sub-standard risks.

GALL BLADDER DISEASE AND GALL STONES.

It has been remarked but recently that "cholecystitis is the most common of all abdominal diseases". The discomfort, fulness, flatulence, vomiting, etc., are often but expressions of an abnormal condition in and about the gall bladder, and many dyspeptic patients particularly those of later years are doubtless gall bladder cases. While much has been written on gall bladder disease it is regrettable that there are yet no criteria immediately available to our examiners by which a diagnosis of cholecystitis may be accurately made. How true this is may be shown by reference to an address on this subject by Dr. Lyons in which he

Medical Aspects of Intestinal Diseases 249

states that of five diagnostic means at our disposal, the *history* is of the greatest importance. We are ready to agree with Dr. Lyons that there are many cases in which the history alone often fails us. In like manner we are in accord with this keen observer regarding the value of the physical examination. This, too, often fails us. If, however, recurring attacks of discomfort or pain in the epigastrium and beneath the right costal margin are reported accompanied by a history of tenderness over the liver at the 9th costal cartilage we have evidence very suggestive of gall bladder disease. Regarding this matter of early diagnosis, however, since failure comes through lack of a clear history or a total absence of physical signs or even a misleading cholecystogram, Dr. Lyons while professing to "refrain from any hard riding" of his biliary tract drainage method regards this as "the only method of diagnosing with some degree of scientific accuracy short of actual tissue study, a first grade cholecystitis of the catarrhal variety". Reports of such studies are but rarely available for life assurance records, and hence figure but seldom in decisions at Head Office.

In discussing the nature of the infection in cholecystitis Professor Wilkie says that "we are thus faced with the inevitable conclusion that chronic cholecystitis is generally a blood borne streptococcal intramural infection and that this micro-organism has a selective affinity for the wall of the gall bladder". This statement of the mode of infection taken with that which declares the bile cultures sterile in so many instances would seem to detract materially from the value to be attached to gall bladder drainage in the *early* diagnosis of cholecystitis.

However, there is some comfort for the Medical Director to be found in the thought that while "cholecystitis is so common an abdominal disease and while diagnostic methods short of biliary tract drainage are often unreliable and a simple inflammation of the gall bladder may recur from time to time with a symptomatology general and severe yet it is seldom that cholecystitis assumes grave importance unassociated with stones in the gall bladder. Gangrene and spreading peritonitis occur in only a small

percentage of cases. The incidence of gall stones is variable. It has been said that 10% of the adult German people are affected. Autopsy records show the same percentage. Combining various European and American necropsy records Mitchell found in 122,808 cases, 7,022 with gall stones, or in 5.7%. (Rolleston & McNee.)

THE LIVER AND PANCREAS IN GALL BLADDER INFECTION.

Whatever may be shown finally as the whole reason that the impairment of the risk increases in proportion to the frequency of the attacks of hepatic colic experienced by the applicant, a large factor in such cases is the spreading and later results of the infection. This is borne out by the advanced pathological changes in the gall bladder wall, the local pericholecystitis and the local or general hepatitis seen either during operation or at necropsy. The infection is blood borne, by the lymphatics or by contiguity. Since cholecystitis is so common among abdominal diseases and since the infection inducing it is blood borne it is not unreasonable to believe that the liver and pancreas may be simultaneously involved. For some special reason it would seem that the gall bladder harbours the infection more favorably than either of these contiguous organs and becomes an established and threatening focus of infection. With gall bladder disease changes of microscopic and sometimes even of gross character are frequent in both liver and pancreas. Dependent on altered functions of one or both of these organs, the gravity of gall bladder cases increases especially when operative measures become necessary. The failure of operations to give relief may be explained by the condition found not infrequently in liver or pancreas.

PANCREATITIS.

Until more definite clinical evidence is to hand regarding the lesser disturbances in this organ our best practice is that of refusing assurance when there is good reason to suspect the pancreas is affected. It would seem that the involvement of this organ is simultaneous in a large proportion of cases (30%) of

gall bladder disease. Whether brought about by infection common to both organs or by obstruction of the bile secondary to stone in the duct or otherwise, is difficult to determine. While a study of the pathology of the living indicates pathologic changes in the pancreas in 25% to 30% of cases, at post mortem this organ is found normal in about 50% of cases.

The spectacular features attributable to pancreatic disease needless to say, are quite readily recognized. Among the diseases referred to, acute pancreatitis, diabetes, and cysts of the pancreas may be mentioned. As already pointed out the minor involvement of this organ during the progress of cholecystitis or cholelithiasis apart from hæmorrhages or the presence of sugar in the urine must be suspected. A comparatively small number of cases has been observed in our office in which there has been an association of gall bladder disease or perhaps operative measures for gastric or duodenal ulcer, with a history of sugar in the urine or glycosuria. The concurrence has been noted but the frequency has not been recorded. Doubtless this concurrence has been observed by many of our members. Lichty and Wood's article on the significance of glycosuria in biliary tract disease was based upon the observation of 23,464 patients, of whom 1,474 showed evidence of biliary tract disease, while 25 of these had glycosuria (not regarded as diabetic).

Regarding the significance of this association the figures were submitted to Raymond Pearl for an opinion. Professor Pearl concluded that there was no basis found for asserting that the association of biliary tract disease with glycosuria is either more or less frequent than would be expected from a chance association. This analysis was made in 1923. Has there been any reason adduced since to alter this opinion?

Dingman points out that pancreatitis due to gall stone obstructing the common duct may be accepted "standard" providing there has been "a year's full recovery after removal of the offending gall-stone". This period of full recovery seems altogether too short even for the single impairment of stone in the common duct with operation for its removal. A much longer time—four

or five years of freedom—have been required by most companies before accepting "standard".

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DR. PATTON—We had hoped to have with us another Canadian representative, or one might say equally as well, another representative Canadian in Dr. Charles F. Martin. Our Secretary received a telegram of regret on October 21 and the statement that important matters would prevent his leaving Montreal.

We are sorry that Dr. John W. Fisher of the Northwestern Mutual could not be with us this year, but we welcome another representative of that company who will continue this discussion. Gastrointestinal diseases are responsible for many of the ills of mankind. They also are the cause of many of our problems in the selection of life insurance risks. I have known Dr. David E. W. Wenstrand since before either of us became connected with the Home Office Medical Department of a life insurance company. I am sure that what he has to tell us will be of real value in our daily work. Dr. Wenstrand.

Discussion—Gastro-intestinal Diseases 253

DR. WENSTRAND—Mr. President and Gentlemen: In a preliminary way, I want to say that I have certainly enjoyed the discussion of the surgical aspects, and in anything that I may have to say it is rather difficult from a life underwriting standpoint to separate the surgical and the medical.

It would be presumptuous on my part to try to add to the thoughts of an eminent clinician and teacher on a subject such as he has covered, but I feel that it is a privilege to be asked to discuss this important topic.

In his introduction, Doctor Hamilton refers to the difficulties presented in the diagnosis of gastro-intestinal disorders, even to the internist who has before him the patient, with a history willingly given and carefully elicited, with a first hand physical examination and with detailed laboratory reports. How much greater then is the problem of the selector of risks who must depend upon secondhand information with incomplete and sometimes misleading information!

That we have been groping for a solution is very apparent from the many papers and addresses which have been presented before this body and before the Medical Section of the American Life Convention during late years, much more so than formerly. All of these have reviewed the knowledge and literature of the subject in a very comprehensive and learned manner, but still, as the essayist says, "This Association has been using comparatively rough methods for many years in assessing values of gastro-intestinal assurance cases," and again: "Our President, in arranging the work of this session, has doubtless been prompted by a desire to get a better expression of opinion concerning digestive tract impairments so that finally a system may be evolved and correct values determined." This is "a consummation devoutly to be wished," and if I can add something from the practice and experience of the department which I represent it will be most gratifying.

Life insurance statistics are sadly lacking in information as to mortality figures in the various groups of gastro-intestinal disorders. To be sure, there were three classes studied in the Specialized Investigation of 1903 and a similar number in the

Medico-Actuarial Investigation, not including fistula in ano, hernias and peptic ulcers, which last named class had too few cases for study. In 1919, Mr. Arthur Hunter compiled some interesting figures from data furnished by the Mayo Clinic of their operated ulcer cases. There have been other studies, as for example the table of indefinite gastro-intestinal disorders given by Doctor Grosvenor in the 1923 Volume of our Transactions, and no doubt some with which I am not familiar.

The Northwestern used the cards returned by the Medico-Actuarial Committee for a compilation and has added to the data, as was reported by Doctor Fisher at the 37th Annual Meeting, in discussing the address of Doctor Frank S. Mathews on the, "Outlook for Life and Health of the Gall Bladder Patient." Perhaps it might be of interest to repeat these figures as far as they relate to the present discussion and to add one item. It may be unfortunate that three Mortality Tables have been used, as the studies were undertaken at different times, but still the results are of definite comparative value.

I. APPENDICITIS WITHOUT OPERATION.
LAST ATTACK WITHIN TEN YEARS.

<i>Years</i>	<i>Policies</i>	<i>Deaths</i>	<i>Mortality M. A. Table %</i>
1885-1908 to 1909	1295	29	77.37%
1906-1915 to 1920	2103	50	<i>A. M. Select Table</i> 67%

II. APPENDICITIS WITH REMOVAL OF APPENDIX.

1885-1908 to 1915	2636	87	<i>O. A. M. Table</i> 36.9%
1901-1908 to 1915	2410	72	<i>A. M. Select Table</i> 62.9%

III. GALL STONES AND BILIARY COLIC.

1885-1908 to 1909	178	15	<i>M. A. Table</i> 136.12%
1885-1908 to 1915	205	23	<i>O. A. M. Table</i> 110.5%
1901-1908 to 1915	115	9	<i>A. M. Select Table</i> 188%

Discussion—Gastro-intestinal Diseases 255

The value of the very small third group rests in the fact that the results are similar to the findings in Class 12 of the Medico-Actuarial Investigation. However, even at that, the class is not entirely homogeneous, and further there has been a remarkable advance in diagnosis and treatment since the data were gathered, so I hesitate to hazard an opinion as to the reliability of the figures at the present time, but it is to be hoped that the investigation now under way will help to clarify many of our doubts.

We sometimes wonder, in our department, why the companies which do a large sub-standard business have not obtained their own experience of such groups, especially as to the various gall bladder conditions and ulcer of the stomach and duodenum, which no doubt would be very informative.

It is not strange then that our action on gastro-intestinal conditions has been individualistic and empirical, largely based upon our medical knowledge and experience. But here again we have, no doubt, often been misled, either intentionally or unintentionally, by the history which has been furnished. For example let me cite a case: In December, 1928, a man 48 years old, Dean of a College of Forestry, was examined, and one of our careful examiners obtained a statement from the family physician in regard to a gastric upset with vomiting a year previously, that the disturbance was due to overeating of rich foods during the holidays and that there was no evidence of organic disease. In every other respect the applicant seemed to be a first class risk, so the policy was issued. He died on September 3, 1929, of what was called a "ruptured gall bladder," an operation of some kind having been done on August 22. In the claim it was said that the abdominal affection had begun two and one-half months previously.

It seems to me then, that if a paper such as Doctor Hamilton's could be widely circulated and thoroughly digested by our examiners, it would really do much more to help us in our problem than all the discussion we might indulge in here. Primarily in all cases it is a question of having all the facts in order to

classify the risk properly, for unless we have the facts, impartially presented in an examination and the resulting correspondence, our knowledge of the theory and practice of risk selection is of no avail. This is especially important in these cases where, as Doctor Hamilton says, "the mists gather and the shadows deepen."

This is hardly the place to refer to the selection and training of examiners, but I cannot refrain from expressing the opinion that this has been and is one of our greatest problems. In a recent address, "On the Diagnostic Importance of Physical Examination," Doctor William S. Thayer, before a gathering of Army Surgeons, said among other things that an army surgeon should have a good knowledge of anatomy, physiology and pathological anatomy including bacteriology, and that he also should have a sufficiently good training and experience in the application of the fundamental methods of physical diagnosis to be able, by means of his five senses, aided by the simpler instruments of precision and those laboratory procedures which are almost always available, to arrive at a reasonable diagnosis himself and to know when and where he needs assistance. This might equally well be said about the life insurance examiner and to it I would add—the ability and the judgment to distinguish between the wheat and the chaff in a given history, thus painting a clear picture of the man before him, so that we may interpret it correctly. That this ideal has a direct bearing on our discussion, I believe is self evident.

For many, many years it has been the practice of our department to get as complete details as possible in regard to all digestive disturbances mentioned in an examination. We write for information from the attending physician, usually through our examiner, asking not only for the probable diagnosis but also for a description of the symptoms and physical findings. We believe that this is important, for a history of the case helps to judge the accuracy of the diagnosis and gives the company the benefit of such doubt as may exist. In our work, as well as in clinical medicine, an individual case often illustrates a point

Discussion—Gastro-intestinal Diseases 257

much better than any amount of abstract discussion, so I hope you will bear with me once more as I recite the following:

In June, a man 33 years old was examined by an experienced examiner in one of the large eastern cities. So called gastric pains of indefinite character were mentioned, which were said to be of no significance. However, we were not satisfied and asked for further details from his physician. Even follow up letters were not answered, so the application was placed on the unfinished file. Late in September, our general agency office notified us that this man had just died of cancer. We can all easily imagine what does not appear on the surface of this story.

In our passing upon this class of risks, after having obtained all possible information, we try to distinguish between acute and chronic conditions as well as between non-organic and organic. We follow such lines as have been outlined in Doctor Hamilton's paper, with the feeling that generally speaking many acute upsets, if not recurrent, are non-organic, while as a rule chronic and organic go together. In the acute, non-organic disorders, we allow a reasonable length of time to elapse before acceptance. We believe that the time element is a very helpful consideration. Where there is a history of one acute attack of appendicitis without operation, the period of postponement is eighteen months. In uncomplicated appendectomies, we sometimes accept the risk as soon as six weeks after complete recovery from the operation.

In connection with the acute gastro-intestinal disturbances, there is particularly one factor in the differential diagnosis, mentioned by Doctor Hamilton, which I should like to stress, namely, angina pectoris. We believe that in writing for information concerning the so-called attacks of acute indigestion in men past middle life, it is necessary to caution the examiner to satisfy himself that there were no anginal symptoms. We all know how often mild and sometimes even severe angina pectoris is called indigestion.

As has already been said, peptic ulcer and gall stones are essentially chronic diseases. We doubt very much that applicants with definite histories of ulcer of the stomach and gall stones,

even after operation, as a rule ever become standard risks; consequently such cases are generally declined. Sometimes, where it is shown that gall stones were removed before middle life and that there has been no recurrence nor sequelae for say about ten years, we have granted insurance. While we have not accepted ulcer of the stomach cases, we issue policies to those who have had ulcers of the duodenum, either with or without operation, provided there has been complete freedom from symptoms for at least five years.

I have nothing to say in regard to other conditions mentioned by Doctor Hamilton, for our experience has been too limited, and we have no definite practice. Cases of chronic colitis of the various types, and of tuberculosis of intestines and peritoneum have been met with so rarely that we feel it is very probable that our agents have done the selecting for us by not submitting applications.

DR. PATTON—The next speaker comes from the Home Office of a member company of our organization that has always been active in our affairs. Dr. Eugene F. Russell has attended our meetings for a number of years and when he joins our discussions he brings us food for thought. We have every reason to feel that what he has to tell us today will add to our ability in the selection of applicants with a digestive history.

DR. RUSSELL—Dr. Patton and Gentlemen: I hope that this will live up to what Dr. Patton has said, but I doubt very much if it will.

Dr. Hamilton has covered the subject of gastro-intestinal tract diseases in such a thorough and satisfactory manner that there is very little, if anything, to add. His paper illustrates very definitely the necessity and importance of paying strict attention to the slightest history referable to the gastro-intestinal tract. With the difficulty experienced by clinicians, when a full history is given freely and willingly, of arriving at a correct diagnosis, it is small wonder that many cases of ulcer, cancer, or gall stones, escape the Examiner and Medical Director. However, the

Discussion—Gastro-intestinal Diseases 259

trained Examiner should be able to form a definite opinion in many cases, and this coupled with the statement from the attending physician, which should be required in all cases giving a recent history, or where a large amount of insurance is involved, should give the Medical Director a diagnosis which, to say the least, is correct in a majority of instances.

It is my feeling that the adverse results experienced in the group of risks is due to several reasons among which is the lack of knowledge as to the importance of trivial symptoms on the part of the Examiner and also the practice on the part of many Examiners to neglect investigating thoroughly the slightest symptoms given by the applicant. Many times a conscientious Examiner has been able to develop, by a few leading questions, gall bladder disease or ulcer from a simple statement made by the applicant.

Dr. Hamilton has summarized the question very well when he states "that the task of our Medical Directors in this connection is to stimulate our Examiners to get more accurate histories, securing all the available data, to keep ourselves fully informed as to the bearing of such data, separating the functional from the organic and, in the light of experience of thousands of cases make our decision accordingly."

With the better understanding that the Examiners have of the importance of gastro-intestinal symptoms and the improvements in the diagnostic methods, the mortality results will be considerably improved.

It may be of interest to the members of this Association to have some of our results presented. The experience of the Mutual Life has been small and is in the course of preparation for the Joint Committee Investigation. In the issues of 1909 to 1927 exposed to 1928, the results of gastric ulcer without operation showed the following results. There were no deaths in the group showing gastric ulcer 5 years previously. From 6 to 10 years previously there were two deaths giving a mortality of 137%—one from suicide and one from undetermined cause. Gastric ulcer with operation, one attack 5 years—mortality of

46.1%, 6 to 10 years mortality 79.2%. There were four deaths, one from cancer and three from other causes. There were no deaths from duodenal ulcer without operation or duodenal ulcer with operation in any of the groups of cases. The groups are too small to arrive at any satisfactory conclusion.

Dr. Hutchinson mentioned something that is very important and which we have taken into consideration in our selection of cases, and that is that the cases that are operated upon must be operated upon by a competent surgeon. We can not take Tom, Dick or Harry as a surgeon in the selection of these cases. Another thing which will give a very definite idea as to how to select these cases is the question of weight, in the cases of persons with or without operation. The gain in weight is an index as to the cure. Of course, you have to eliminate the possibility of forced feeding, but if you take a steady gain in weight over a period of years, in which we postpone these cases, the prognosis is good in those cases which have gained weight and which have come up to the standard weight and which hold the weight.

It is interesting to note that out of 186 cases declined between 1918 and 1922 because of the history of gastric ulcer without operation fourteen have died in the interim the causes of death being:

Pneumonia	2
Cerebral Hemorrhage	3
Erysipelas	2
Cancer	1
Appendicitis	1
Intestinal Obstruction	1
Influenza	1
Other Causes	3

Out of 102 cases declined in the same period because of a history of gastric ulcer with operation, 11 have died in the interim, the causes of death being as follows:

Discussion—Gastro-intestinal Diseases 261

Cerebral Hemorrhage	1
Diseases of Heart	1
Diabetes	1
Accident	2
Ulcer of Stomach	1
Pneumonia	1
Other Diseases of Stomach	1
Nephritis	1
Other Causes	2

You will note that there was only one death in this group of cases from cancer and that fact illustrates to some extent the lack of association between cancer and ulcer of the stomach.

Our experience with gall bladder cases has been somewhat larger than that with ulcer of the stomach or duodenum. Gall stones without operation, one attack within two years, shows a mortality of 136.5%, one attack within 3, 4 or 5 years 135%, one attack within 6 to 10 years 125%, two or more attacks last within two years or more 136%. Among this group there were 86 deaths as follows:

Accident	11
Influenza	3
Tuberculosis	2
Cerebral Hemorrhage	7
Diseases of Heart	6
Cancer	5
Pneumonia	4
Nephritis	3
Suicide	7
Diabetes	1
Appendicitis	1
Other Causes	36

Gall stones with gall bladder removal, one attack within two years 124%, one attack within 3, 4, or 5 years 119%, one attack within 6 to 10 years 73.3%. There were 26 deaths in this group as follows:

Accident	7
Diseases of Heart	4
Cancer	1
Cerebral Hemorrhage	1
Nephritis	1
Pneumonia	1
Other Causes	11

Gall stones and gall bladder drainage, one attack within two or more years 109% in which there were 33 deaths as follows:

Accident	4
Cancer	5
Nephritis	7
Cerebral Hemorrhage	4
Suicide	2
Influenza	1
Diseases of Heart	1
Other Causes	9

You will note that cancer occurs quite frequently among the causes of death where there is a history of gall stone or gall stone operation, whereas cancer deaths are below the expected in ulcer of the stomach. Among 206 cases which were declined because of a history of gall stones between 1918 and 1922, 22 deaths occurred as follows:

Anaemia	1
Cerebral Hemorrhage	4
Embolism	1
Organic Disease of Heart	1
Other Diseases of Liver	1
Diseases of Arteries	1
Pneumonia	1
Angina	1
Accident	1
Cancer	2
Influenza	1
Not Specified	7

Of 52 cases declined where gall stones or gall bladder was removed there were nine deaths:

Cancer	2
Diseases of Heart	2
Diseases of Arteries	1
Nephritis	1
Cerebral Hemorrhage	1
Other Diseases of the Circulatory System	1
Not Specified	1

Of 70 cases declined with a history of gall stones and gall bladder drainage there were fifteen deaths.

Discussion—Gastro-intestinal Diseases 263

Cerebral Hemorrhage	3
Angina	1
Tuberculosis	1
Nephritis	2
Ulcer of Stomach	1
Suicide	1
Intestinal Obstruction	1
Other Diseases of the Respiratory	
System	1
Broncho Pneumonia	1
Alcoholism	1
Cancer	1
Not Specified	1

There is nothing further to be said about achylia gastrica or tubercular peritonitis as these cases are few and far between and fall into the category of experimental insurance. A word about pancreatitis. We should all recognize the fact that with almost every case of gall bladder disease there is more or less associated chronic pancreatitis and that when the cause is removed it takes a considerable period of time for the pancreatic tissue to adjust itself and that the slightest indication of deranged digestion or the slightest presence of sugar following gall bladder disease is, in the majority of instances an indication that there is some chronic inflammation present in all probability in the pancreas and should be treated accordingly.

DR. PATTON—The medical departments of life insurance companies have availed themselves of the advances in medical and surgical and laboratory methods. None of our companies have shown greater activity in this direction than the one with which the next speaker is connected. Those of us who were present last year recall with pleasure the initial appearance in our discussions of Dr. Haynes H. Fellows of the Metropolitan Life, and we hope that he will bring to us some of the new thoughts of the present day as to some of the causative factors of digestive disorders and some of the constitutional effects of such conditions. Dr. Fellows.

DR. FELLOWS—When one approaches the subject of disease of the gastrointestinal tract in applicants for life insurance, there are a few outstanding facts evident at once. In the first place, any physician who has had experience in treating patients knows

the minute, painstaking history which must be obtained with the full cooperation of the patient before any clew as to the probable source or cause of symptoms can be obtained, and this is especially true when dealing with gastrointestinal cases. After some idea is gained as to where to search for a possible lesion, the next step necessary is a careful laboratory examination, almost always including an X-ray examination of the gastrointestinal tract and the gall-bladder, together with special laboratory tests, including gastric chemistry, examination of bile obtained by biliary drainage, blood examination including serology, proctoscopic or rectal examination, all of which again can be obtained only with the full consent of the individual.

When such an examination is carried out, and only then, we know that a diagnosis can be made with approximately 95% degree of accuracy. This is the accepted standard among such institutions as the Cornell Clinic, the Lexington Clinic of Kentucky, the Mayo Clinic, and others. So much for what *can* be done.

Now, let us consider the subject of gastrointestinal symptoms and disease as reported at the time of life insurance examinations. Upon 168,000 issues and rejections of the Metropolitan Life Insurance Company in 1922 and 1923, gastrointestinal symptoms were admitted by only 6.5% of all of the applicants. An history of appendicitis, with or without operation, was admitted by 4.3% of the applicants. So that only 2.2% of this group gave a history of any other diseases of the gastrointestinal tract and its allied organs which have been elaborated upon so well by Dr. Hamilton. It is perfectly apparent that this small percentage of positive histories does not give the true picture of these 168,000 people, but can we find in any way what this picture should be in this group of applicants?

A thorough, conscientious search of the medical literature of the world does not tell us anything about the percentage of gastrointestinal disease to be expected in a cross section of the average population. Hospital autopsy findings obviously cannot be used, as only the sick go to hospitals. Necropsy findings

on persons dying from accident would be valuable in discovering the incidence of gastrointestinal as well as other disease and I find that data are available in several of the larger cities, but none of these data have been analyzed. Prof. Raymond Pearl at Johns Hopkins has a mass of material, some of it of this type, but so far it has not been published. Findings on examination by the Life Extension Institute give some indication of the number of histories of gastrointestinal disturbances that we might expect in a group of persons at work. In a survey of 10,000 supposedly well employees in more than 100 industrial plants, the Life Extension Institute in its health examinations found a history of definite digestive disturbance in 29% of the cases. In a group of approximately 17,000 male policyholders in the Metropolitan Life Insurance Company, *18% gave a history of gastrointestinal or severe digestive disturbance when examined by the Life Extension Institute. Of course, it is possible that an undue proportion of those who availed themselves of the opportunity of taking this Life Extension Institute examination offered to policyholders were not in the best of health and this may be one reason for the high percentage of gastric symptoms. No doubt, too, many more gave a more complete and proper answer to these questions than to those asked at the time when life insurance was applied for. However, the difficulty experienced in securing a proper history of the gastrointestinal troubles is the same as is met in the endeavor to get correct histories of all illnesses and symptoms of applicants for life insurance.

I don't believe that anyone would argue that the applications for life insurance give a true picture of the applicants from a medical standpoint. The medical examiner fails very frequently to follow up leads given by the applicant regarding important points in the history, and probably more frequently the applicant makes no mention of symptoms which are or have been present and which he feels may cause the insurance to be rated up or not issued. It is true that full cooperation cannot be counted on in many applicants

*"Physical Defects as Revealed by Periodic Health Examinations" by Dublin, Fisk, and Kopf.

and this makes it imperative to assemble concrete facts regarding the case. This is being done already in a rather groping, tentative way, and into the Medical Division of each of our companies are coming daily X-rays, electrocardiographic tracings, special laboratory reports for consideration.

I believe that investigation will bear me out that the study and practice of medicine has progressed to the point where there are located conveniently throughout this country either hospitals, or laboratories, or diagnostic groups, or individual physicians, who if they used all means readily available, could give us more accurate facts than we are obtaining at the present time. These facts I believe should include more concrete information, and by concrete information I would suggest X-ray films, electrocardiographic tracings, and other laboratory data. If all of the life insurance companies would by agreement designate certain such conveniently located laboratories and insist that as a routine the applicants for large amounts be examined more thoroughly, I am sure the medical selection would be made on a better basis of fact and judgment.

Thirty-three cases, each having \$200,000 or more of insurance issued by the companies as a whole in which death occurred within five years, have been studied. Upon two of these cases representing \$510,000 of insurance there was very poor medical underwriting; one individual having had a history of long suspected tuberculosis, as well as a family history of tuberculosis. This man was taken ill 3 months after issue and it was found then that an extensive latent pulmonary tuberculosis had become active. Death occurred within 8 months of the onset of symptoms as the culmination of a severe systemic tubercular infection. The other case gave history of the passing of gravel, pyuria and other genito-urinary symptoms during the year prior to application, and died one year after issue following an operation for bilateral renal calculi. An X-ray examination could have disclosed the condition present in each case. Eight cases representing insurance of \$5,000,000 died of disease of a type which could have been disclosed by an X-ray of the gastrointestinal

tract or in which an electrocardiogram might very well have disclosed the pathological changes. The remaining twenty-three cases died of conditions which probably could not have been detected at the time of application.

The financial saving which could have been effected if proper medical evidence had been available and had been used in this selection would have paid for a complete and thorough examination of thousands of other applicants, as this type of examination is not by necessity prohibitively expensive.

Now apparently more complete knowledge of the case does not imply of necessity a *higher* rejection rate, but a more accurate action. We have had an example of that brought very forcibly to our attention in the last two years at the Metropolitan Life Insurance Company. In October, 1927, we began to examine fluoroscopically the chests of all applicants for employment in an endeavor to detect pulmonary disease. Up until that time our recommendations for rejections because of lung disease were based on a careful physical examination and the rejection rate was quite stable, about 2%. It was thought by some at our Home Office that if this new routine were followed that our lung rejection rate would mount considerably, but it was decided to try the experiment. As a result, in the last two years we have detected among our prospective employees a very considerable number of cases of definite pulmonary tuberculosis which did not give physical signs. We have been able to follow many of these persons for $1\frac{1}{2}$ years and have found that in over 60% of those so followed there has been a marked and definite progression of their disease. Now they are rejected, but in former years we would have employed them. Practically all of those who had been rejected formerly upon physical findings such as harsh breathing, transitory rales, and other vague abnormal signs, showed no lesions by X-ray and most of these are being accepted and so far our experience warrants a continuation of this policy. As a result, we find that our rejection rate is practically unchanged, and our tuberculosis problem is nearer solution.

It is, I suppose, the policy of each company to request certain applicants for insurance who present suspicious or questionable histories or physical findings to come to its Home Office for a more thorough examination. However, since it is practical to reach only such individuals as are located convenient to the Home Office or its various larger branches, only a relatively small number of such applicants can be studied carefully. We at the Metropolitan do exactly this same thing and I am reporting to you upon groups of applicants who were so examined, and am giving you our disposition of the cases after they were more thoroughly studied.

There were selected at random the applications for insurance of 74 individuals upon whom it was necessary to perform special tests or procedures before final action in regard to their application could be taken.

Thirty-three applicants were given a sugar tolerance test: twenty of these cases had a history or M.I.B. record of glycosuria and thirteen showed sugar in the urine at the first examination in sufficient quantity to necessitate a sugar tolerance test. After the grape sugar test was completed and reported upon, there was issued standard insurance to seventeen, substandard to four, and only twelve were declined.

The remaining forty-one cases were given a complete physical examination at the Home Office and also an X-ray examination. Upon twenty of these there was an M.I.B. history regarding the lungs—suspicious tuberculosis or bronchitis,—on seven the field examiner found suspicious signs in heart or lungs by physical examinations, and on the remaining fourteen cases there was some other history or finding on physical examination which made a more careful work-up desirable. In over half of this group the final decision was more favorable than it would have been without the Home Office review, in eighteen it was unchanged, and in only one case was there a rejection where there would have been issued standard insurance.

I have mentioned these experiences to allay the possible fear of those who are responsible for the production of business that

Tuberculosis of Gastro-intestinal Tract 269

a more careful examination will interfere materially with the placing of life insurance. Though some undesirable policyholders undoubtedly will be lost to the Company, other insurable applicants will be saved and a more nearly accurate judgment as regards a fair rating can be made.

DR. PATTON—The inter-relationship between tuberculosis and the digestive tract has always been interesting. Whether tuberculosis precedes or follows the gastrointestinal symptoms is an important consideration, but the gravest feature for us is that if they combine in a policyholder, we are more likely to have to pay an early claim. The consideration of these conditions is brought to us by one of our Canadian brethren who is qualified by both training and practice to discuss the subject, Dr. Jerome F. Honsberger of the Mutual Life Assurance Company of Canada.

DR. HONSBERGER—Dr. Patton and Gentlemen: You will pardon me, I am sure, when I tell you that my paper is somewhat condensed and not lengthy and I may therefore take the liberty of confining myself pretty closely to the text. I am hoping, however, that the discussion arising from this paper may bring out many points which I have purposely omitted on account of my desire to condense my paper.

TUBERCULOSIS OF THE GASTRO-INTESTINAL TRACT.

J. F. HONSBERGER, M. D.

*Medical Director**The Mutual Life Assurance Company of Canada.*

In the preparation of this paper I was fully cognizant of the fact that on account of the difficulty of dissociating tuberculous disease of the alimentary system from tuberculous disease in other parts of the body, particularly of the lungs, statistical material would not be available.

In my search for information on the subject I have succeeded in obtaining a large amount of reliable material drawn from the experience of no less than forty of the largest Sanatoria in the United States and Canada, as well as the opinions of an equal number of the most outstanding surgeons and consultants in medicine, and clinics of international reputation.

From all these sources I shall therefore endeavor to give what would appear to be the consensus of opinion and the generally accepted view of these authorities on the subject under review.

All are agreed that symptoms suggestive of gastro-intestinal disturbance are frequently present in the early stages of tuberculous disease, and that these symptoms may precede any clinical evidence of disease elsewhere.

These symptoms do not necessarily represent a condition of ulceration in the gastro-intestinal tract, although this may be present, but would appear to be functional in character, without any reason to suspect tuberculous disease anywhere in the body.

Dr. I. C. Molony of the Fort Qu'Appelle Sanatorium, Saskatchewan, has contributed an excellent and very interesting paper, being a study of one thousand cases admitted to that institution during a period of four years. At time of admission a complete history of each case was obtained, and particular emphasis was placed on the "entrance complaints" given by the patient, without any suggestive or leading questions. One hundred

Tuberculosis of Gastro-intestinal Tract 271

and ninety-two of the one thousand, or 19.2% gave no complaint referable to the chest but sought advice or treatment on account of abdominal disturbance, anorexia, gastric pain and discomfort, indigestion, slight diarrhoea or constipation. This group of symptoms held fourth place in order of frequency.

He found also that during the period of treatment of their pulmonary cases abdominal disorders covered five out of ten of the symptoms most frequently complained of by the patients.

Of the pulmonary cases 14.3% made no complaint referable to the chest.

Only a small proportion of cases having gastro-intestinal symptoms in the early stage really have any tuberculous lesion in the alimentary canal, but their symptoms appear to be functional in character, and may be an indication of tuberculous disease elsewhere, more particularly in the lungs.

When gastro-intestinal symptoms are present as the result of abdominal disease it is most frequently found to be tuberculous peritonitis when symptoms occur early in the history of the patient; if later, ulceration of the intestines is most frequent.

When Sir William Osler in a public address in England some years ago made the startling statement that ninety per cent of the Anglo-Saxon race had at some time in their lives been infected by tubercle bacilli, the medical profession were inclined to disbelieve the accuracy of the statement. He supplemented this statement, however, by saying that "Evidence of infection is, however, not always available and can only be determined by autopsy".

Modern methods and appliances now used as a means of diagnosis have greatly assisted us, and the statement is now regarded as well within the truth. The German aphorism, "Jederman hat doch ein bisschen Tuberculose" fairly represents the opinion of the profession today.

The tubercle bacillus is admitted into the system by inhalation into the lungs, this being by far the most frequent method of admission. To a lesser degree it may be admitted by means of food containing bacilli. The latter method is particularly appli-

cable in the case of children fed with milk from cows suffering with bovine tuberculosis.

In childhood the presence of tuberculous infection may be fairly accurately determined by means of the Tuberculin Test; but, unfortunately, this test is of little value as age advances and is rarely employed beyond the age of twelve years, being less suitable in adults.

Secondary lesions may occur in the stomach or intestines through the medium of the tuberculous sputum swallowed, or by blood infection through the mucous membranes.

Tuberculosis of the œsophagus and stomach occurs so rarely that it may be considered a pathological curiosity. When it does occur it is usually of the acute miliary type. The acid contents of the stomach are probably responsible for its absence in the stomach and upper part of the duodenum. (Albutt & Rollison, Vol. II, Page 270.)

Asch fed rabbits with tubercle bacilli and obtained intestinal tuberculosis but only once succeeded in infecting the stomach. In one hundred and eight autopsies of phthisical subjects only one showed a lesion of the stomach. Jacobi says that the blood vessels having absorbed tubercle bacilli from the primarily infected part are liable to distribute these in the form of acute miliary tuberculosis. Koch has proven that active bacilli pass the stomach unmolested and may infect the intestine, causing a primary tuberculous lesion.

The most frequent site of tuberculous disease of the intestinal tract is the ileum in the vicinity of *Peyer's Patches*. It is not so frequently found in the large as the small intestine, and its existence there is usually in the ulcerative stage. It first appears as small tubercular nodules which tend to coalesce and caseate, finally sloughing by necrosis, leaving an ulcer. These ulcers are usually irregular and transverse to the bowel, with thickened edges and uneven base, showing small caseated nodules which may be situated in either the muscular or mucous coat. Perforation of the ulcer into the peritoneal cavity is usually prevented by adhesions.

Tuberculosis of Gastro-intestinal Tract 273

Stricture of the intestines may result from contraction of the scar of a healed ulcer. The disease is rarely primary in origin.

Louis laid it down as a rule that if tuberculosis is found after fifteen years of age in any part of the body it is present also in the lungs.

At the Munich Pathological Institute primary intestinal tuberculosis was found in only one case in one thousand autopsies.

Tuberculous peritonitis is insidious in its onset and progresses slowly. The symptoms are moderate pain, rapid pulse, progressive emaciation, anæmia and general debility. In its chronic form loss of appetite, moderate diarrhoea, loss of weight, failing strength, enlargement of the abdomen and occasional pigmentation of the skin are noticeable. Occasionally a mass resembling a tumor may be present immediately above or below the umbilicus. (Greene's Medical Diagnosis.) Fluid is present in a fair proportion of cases. The focus or origin may be a lymph gland, lungs or intestines. Cases in which the disease arises primarily in the peritoneum are so rare that one must question the accuracy of the observation. Tuberculous peritonitis is most common before forty years of age, generally between thirty and forty, but frequently found in younger persons. If re-infection from a focus could be prevented by adhesion or scar formation a fair chance of radical cure might be obtained.

The appendix is rarely affected. Combined statistics of various operators show that in every hundred cases of appendicitis one or two are tuberculous. The bacilli reach the appendix (1) by the peritoneum; (2) by the lymphatics; (3) by the blood.

The liver and spleen are somewhat more frequently affected than the stomach, and here the disease is of the miliary type, and is secondary to a focus elsewhere.

Experiments have proven that if virulent tuberculous material is fed to guinea pigs lesions have appeared in nine days and less rarely in eighteen to twenty-one days.

Infection of the tissues around the rectum gives rise to the variety commonly known as fistula-in-ano. It is, however, not uncommon to find the lower part of the bowel free from ulcera-

tion, although fistula is quite common when extensive ulceration of the lower bowel is present.

That mortality is increased by this disease is shown by Medico-Actuarial Investigation made by forty-three companies from 1885 to 1908 in cases having a history of a single attack of fistula with or without operation. Within two years of application the mortality was 120% of the normal expectancy; from two to five years the mortality was 136%; from five to ten years it was 98% to 100%. Of these a group which were 25% or more overweight gave a mortality little if any in excess of the mortality arising from excess weight alone. A group 15% or more underweight gave a mortality of 113%.

There is a wide divergence of opinion as to the proportion of fistulæ which are tuberculous in origin. Gant gives a record of five hundred cases in which less than 10% were tuberculous and 95% of those had a tuberculous lesion elsewhere. Dr. Stewart of Ninette, Manitoba, says, "The condition seems to have a definite relation to tuberculous enteritis". Dr. Leslie of River Glade, N. B., says, "The majority of our ischio-rectal cases have been associated with enteritis".

Most authorities are agreed that only about 2 or 3 per cent of TB. patients suffer from fistula-in-ano, although many Sanatoria report a higher or lower percentage. Fort Qu'Appelle, Sask., had only five fistulæ in 1735 cases covering a five year period. Ninette, Man., showed 2½ per cent in 850 cases during three years. Tranquille, B. C., showed 42 per cent of 167 advanced cases. While it is true that only a small proportion of TB. sufferers have fistulæ, it is equally true that a large proportion of fistulæ are tuberculous. As the disease generally occurs as a secondary lesion it is highly important that every effort should be made to locate the focus from which infection has arisen.

Dr. C. H. Mayo says, "I recall a few cases of tuberculosis in which the first evidence of disease was a small ischio-rectal abscess".

Dr. Landis of Phipps Institute says, in *International Clinics*, Vol. II, page 1645, "Far from being a simple local condition fistula-in-ano is a distinct warning that pulmonary trouble is al-

Tuberculosis of Gastro-intestinal Tract 275

ready present and the patient should be subjected to the necessary examination to determine the presence or absence of pulmonary trouble".

Dr. David Nussman refers to the fact not to be overlooked that "A tuberculous focus anywhere in the body far from being an isolated affair is but the visible evidence of a systemic infection and in all certainty one of the links in a chain of scattered foci even though we should at the time be unable to place our finger on it".

Evidence may be obtained in various ways: (1) The presence of tubercle bacilli may be demonstrated by inoculation into animals; (2) By histological section; (3) By clinical evidence of disease elsewhere.

Gabriel prefers animal inoculation. In his investigation he found bacilli relatively scarce in the tissues and hard to detect by histological section, but inoculation in animals furnished positive proof in the same cases in which section had failed to show the presence of disease.

The treatment is surgical by excision. Curettage has resulted in many failures. Radical cure has proven successful except when the fistula is secondary to a tuberculous lesion elsewhere. X-Ray therapy is useful as an adjunct in treatment after the acute stage of a rectal abscess has subsided.

The diagnosis of tuberculous disease of the alimentary tract is extremely difficult. Physical signs and clinical evidences are uncertain. Diagnosis was formerly based on the presence of disease in another part of the body. If pulmonary tuberculosis was present and the patient developed abdominal symptoms a diagnosis of intestinal tuberculosis was promptly made and a fatal prognosis was almost invariably given. Many errors occurred. In 452 cases shown by autopsy to have tuberculous lesions of the intestine Kieffer had not been able to diagnose this condition in more than 250 cases. Walsh of the Phipps Institute from his observation concluded that it is impossible to diagnose the presence or absence of tuberculous intestinal disease.

A marked advance was made about a decade ago by the introduction of the barium meal and Roentgen ray test. The

marked hypermotility as shown by this test in cases of intestinal ulceration gave almost positive evidence of disease. The character of the ulcer can, however, not be distinguished from typhoid ulcer, but in this respect clinical evidence gives strong support to the diagnosis arrived at. The barium meal with Roentgen ray examination is now recognized as a reliable test and is extensively employed by sanatoria and the profession generally. Dr. Lawrason Brown of Saranac Lake and Dr. Stewart of Ninette in recent publications have strongly advocated its use and are using it constantly in their respective sanatoria.

The treatment of the intestinal type of this disease does not differ from that of disease elsewhere excepting that some modified treatment must be employed for local symptoms, e.g., diarrhoea. As in pulmonary disease, sanatorium treatment is most desirable and gives best results. Surgical treatment is useful if the lesion is sufficiently localized to warrant its removal, and excellent results have followed in many cases. Surgery has given fairly satisfactory results in suitable cases, but heliotherapy is a valuable adjunct and is now an approved method of treatment.

The prognosis in all forms of intestinal disease is dependent very largely on the disease being primary or secondary. If the general disease has advanced beyond the incipient stage and the intestinal lesion is secondary the prognosis is not favorable. Tuberculous peritonitis is rather more favorable and many cases have recovered.

The question of danger from contagion constantly arises. If the disease is confined to the alimentary canal there is little if any danger, unless the patient lives in a rural district where the modern sanitary conveniences are not available. There is no guarantee, however, that the lesion which may be primary may not be a focus for further infection, and future segregation must be assured if exposure is to be disregarded.

Blood invasion of TB. bacilli is very common in the intestinal type and reinfection frequently occurs in lung cases through the lymph and blood.

Tuberculosis of Gastro-intestinal Tract 277

The question of insurability of those suffering from or having a history of tuberculous disease of the gastro-intestinal system naturally arises in a review of this subject. The same rules or conditions which govern in pulmonary tuberculosis may be applied here, after a most careful investigation by approved methods of diagnosis, such as barium meal and Roentgen ray, have been employed.

An applicant giving a history of healed fistula-in-ano may be accepted providing build and family history are favorable and inoculation has not shown the lesion to have been tuberculous; providing also that clinical evidence and X-ray of the chest are negative.

If such thorough investigation has not been made a period of two years should elapse before acceptance.

If fistula be present at time of examination the case should be declined.

Dr. Wodehouse, Secretary of the Canadian Tuberculosis Association, has furnished us with some interesting data concerning the ratio of deaths from intestinal and peritoneal tuberculosis to that of the total deaths from tuberculosis in all forms in Canada from 1922 to 1927 inclusive.

TUBERCULOSIS OF THE INTESTINES AND PERITONEUM.

Year	Population of Canada Except Quebec	Rate per 1000 Total Deaths All Causes	Total Deaths All Forms Tuberculosis	Tuberculosis Death Rate Per 100,000 Population	Total Deaths TB. of the Intestines & Peritoneum		Percentage TB. of Intestines & Peritoneum of Deaths All Forms TB.
					Male	Female	
1922	6,552,662	68,028 10.5	4633	71.0	89	216 127	4.7
1923	6,691,273	70,182 10.6	4800	71.0	72	201 129	4.3
1924	6,735,000	66,197 9.8	4647	69.0	72	178 106	3.8
1925	6,832,000	66,477 9.9	4531	66.0	67	146 79	3.2
1926	Including Quebec 9,378,000	107,464 11.45	7930	84.5	135	334 199	4.3
1927	9,507,000	106,392 11.1	7764	81.7	156	540 184	4.4

It may be of interest to note that during the years 1922 to 1925, inclusive, the mortality from all causes in Canada, excluding the Province of Quebec, was 9.9 per 1000. Previous to the year 1926 figures for the Province of Quebec were not available on account of there being no registration in that Province. During the years 1926 and 1927 Quebec was included, and the mortality rose to 11.45 per 1000, being an increase of 14 per cent, and the death rate from tuberculosis rose from 66 per 100,000 to 84.5, being an increase of over 25 per cent; also that whilst the percentage of deaths from tuberculous disease of intestines and peritoneum in Canada decreased previous to 1926, it began to increase after Quebec was included.

In conclusion I desire to express my appreciation of the assistance given me in the preparation of this paper by Dr. Coutts, of the Freeport Sanatorium, and many others who so willingly furnished me with information, as well as the assistance afforded me by the reading of "Intestinal Tuberculosis," by Dr. Lawrason Brown and Homer L. Sampson, of Trudeau Sanatorium, Saranac Lake.

DR. PATTON—Dr. Ward will open the discussion.

DR. WARD—Doctor Honsberger's study of Tuberculosis of the Gastro-Intestinal Tract shows extensive research and supplies some helpful suggestions for medical selection. He emphasizes the very important fact that in a large proportion of tubercular cases the early symptoms are disturbances of digestion rather than pulmonary symptoms. Furthermore he shows that in only a small proportion of these cases can actual lesions of the gastro-intestinal tract be demonstrated and that in a large proportion of cases these disturbances of digestion are entirely functional in character. He further shows that tuberculosis of the stomach is a rare condition and that when it does occur it is in connection with a diffuse miliary tuberculosis. The most common site of tubercular lesions of the intestines is in the vicinity of Peyer's Patches.

Referring to fistula in ano he states that authorities agree that from two to three per cent of all tubercular cases are

Discussion—T.B. of Gastro-intestinal Tract 279

complicated with fistula, but he adds the valuable warning that a patient with fistula should be subjected to such examinations as may be necessary to determine the presence or absence of tuberculosis.

From an insurance view point the most valuable suggestion seems to be a more careful analysis of applicants applying for insurance who present disturbances of digestion. We should not be satisfied in excluding disease of the appendix, the gall bladder or the duodenum but we should bear in mind that frequently these symptoms are indications of tubercular disease of some part of the body and we should give particular attention to the chest examination. Particularly is this true if these symptoms are accompanied with fatigue, any abnormality of temperature or a low blood pressure.

As to fistula in ano this same thought should be present. If the fistula occurs in an apparently vigorous individual, if it be unaccompanied with any symptoms suggestive of tuberculosis, if the applicant be of good physique and especially if an operation is followed by a rapid healing then we may with reasonable safety accept such risks as standard. On the other hand the recurring fistula or the fistula that heals slowly should arouse our apprehensions.

It may be of interest to submit a few figures showing the decline of deaths for tuberculosis of all types in our Company's statistics. The Mutual Benefit was organized in 1845. Fortunately we have accurate mortality statistics from that date.

From 1845 to 1858 the deaths from tuberculosis constituted 18.47% of all deaths. From 1858 to 1865, 19%. From 1865 to 1870, 19%. From 1845 to 1870 there was no improvement in the mortality from tuberculosis the proportion being 19% of all deaths. From 1870 to the present time the decrease has been progressive as the following figures demonstrate—

1845 to 1858	18.4%
1858 to 1865	19.0%
1865 to 1870	19.0%
1870 to 1875	15.3%
1875 to 1880	12.2%
1880 to 1885	11.6%
1885 to 1890	10.7%
1890 to 1895	8.2%
1895 to 1900	8.9%
1900 to 1905	8.7%
1905 to 1910	8.2%
1910 to 1915	7.2%
1915 to 1920	6.4%
1920 to 1925	5.8%
1925 to 1928 inc.	4.6%

These figures demonstrate the great accomplishment in controlling the ravages of this disease. It is a satisfaction to know that the Life Insurance Companies by their publicity on hygiene and their emphasis upon periodic examinations have contributed materially to this result.

DR. PATTON—Some years ago it was my good fortune to meet Dr. Charles A. VanDervoort and subsequent meetings have served to increase my respect for his knowledge of our work. I therefore am pleased to call upon him to discuss the subject of Tuberculosis and Diseases of the Gastro-intestinal Tract. Dr. VanDervoort.

DR. VANDERVOORT—Our esteemed President, Dr. Patton, asked me to confine my discussion of Dr. Honsberger's paper to the possible influence of gastro-intestinal disorders on the development of tuberculosis in other parts of the body, especially in the lungs.

Because of the nature of my task, my discussion must of necessity be along negative lines; an attempt to demonstrate that up to date we have not been able to prove conclusively that pulmonary tuberculosis is due, or can be due directly to gastro-intestinal disorders.

I have searched the literature diligently; I have consulted a goodly number of internists and gastro-enterologists, and have not succeeded in finding any positive proof. However, there is general agreement that prolonged digestive disorders leading

Discussion—T.B. of Gastro-intestinal Tract 281

to anæmia, malnutrition and loss in weight surely predispose to pulmonary tuberculosis by wearing down resisting power.

The tubercle bacillus, an organism of much determination, does not hesitate to attack a potential victim, whether he be healthy or sickly, strong or weak, young or old. So far as we can tell its attacking power is approximately the same in degree, no matter what or where the victim. Whether or not its baneful influence be successful depends entirely on the fertility of the soil in which it is planted. Hence an organ or tissue, weakened by hereditary disease, local disease or trauma, or lack of properly oxygenated blood supply becomes a suitable field for the bacillus.

According to the literature the stomach is exceedingly resistant to primary infection by the tubercle bacillus. This is presumed to be due to its vascularity, its motility and to the character of its secretions.

Broders reports that "in 2501 operations on the stomach at the Mayo Clinic, but one case of gastric tuberculosis was found," and further "that the relative number of specific tubercular lesions is slight even in cases of pulmonary tuberculosis."

Musser in 1890 was the first in America to demonstrate tubercular gastric ulcer. Herz says that "less than one percent of tuberculous individuals show a stomach lesion, and then only as a terminal manifestation."

We must then first of all, I believe, concede that primary tuberculosis of the stomach is an exceedingly rare condition. Again, is it not astonishing that the stomach is so rarely infected directly, when we realize that bacilli laden sputum is swallowed in quantities by those suffering from pulmonary diseases?

There is evidence in abundance to prove that gastro-intestinal disorder is frequently a very early and a very significant symptom of incipient pulmonary tuberculosis.

Dr. Honsberger has directed our attention to two very significant facts,—“that symptoms of gastro-intestinal disturbance are frequently present in the early stages of tuberculous disease and that they (gastro-intestinal symptoms) may precede clinical evidence of disease elsewhere” and that “19.2% of the patients

admitted to the Fort Qu'Appelle Sanatorium sought advice on account of gastro-intestinal symptoms, but gave no complaint referable to the chest."

Let me quote a few other authors.

Musser: "Diseases of the stomach frequently mask other diseases, one of them tuberculosis, and is entirely due to the primary disease. In tuberculosis the local gastric symptoms are often the most pronounced feature."

Kemp: Speaking on intestinal tuberculosis—"The condition may begin with irregular diarrhea, fever and colicky pains. At first the symptoms may simulate chronic catarrh and until emaciation becomes marked or the lungs involved the condition may not be suspected."

Klebs: (1909) "A large number of cases of pulmonary tuberculosis begin as a dyspepsia and few fail sooner or later to show some evidence of gastric disorder."

Jnowski: (1907) Found gastric disturbance in fourteen per cent of seven hundred incipient cases of pulmonary tuberculosis.

Behring: (1904) Regards this lack of good appetite as an early symptom of the presence of tuberculosis somewhere in the system. It could usually be considered rather as an excellent predisposing cause through its depressing effect on the general nutrition.

"Fermentative dyspepsia is a very common complaint of tuberculous patients and is often seen very early."

Rehfuss: (1927) "While true gastric tuberculosis in the form of an ulcer, miliary tuberculosis or a diffuse process is rare, a disturbance of gastric function is almost constant in tuberculosis of the lungs."

Conceding, therefore, as I believe we must, that pulmonary tuberculosis is not directly due to primary disturbance of the gastro-intestinal tract, may there be a method by means of which the latter may be an indirect cause.

It is conceivable to presume that a great many pathological conditions lay the ground work and prepare the soil for future tuberculous invasion. Anæmia, while an associated condition with tuberculosis, may primarily lower the vitality so as to

Discussion—T.B. of Gastro-intestinal Tract 283

permit invasion to occur and rapidly spread. The coincidence of pernicious anæmia and tuberculosis is well known. Many cases of influenza, especially in subjects not particularly robust, terminate in pulmonary tuberculosis. An acute poorly cared for bronchitis may terminate likewise. Why not then believe that a person, harassed and tormented for a long time by so-called indigestion, is a good subject for tuberculosis? The difficulty is, as pointed out by Dr. Honsberger and many others, that while digestive symptoms are present in such a large proportion of cases of incipient tuberculosis and is the complex for which the patient seeks advice, without the slightest reference to lung symptoms, we do not know the definite relation which exists between the two as primary factors. Did the digestive organs suffer first and by lowering vitality predispose to tubercular invasion, or, on the other hand, did the latter, in its very earliest stages, exert an evil influence on the former? Either, I think, may be correct; but I believe definite tubercular pathology in the lungs, even without subjective symptoms, actually precedes the onset of gastro-intestinal symptoms.

SUMMARY: A.—Gastro-intestinal disorder as a definite cause of pulmonary tuberculosis, so far as our present knowledge is concerned, must be excluded.

B.—Gastro-intestinal disorder, by lowering resistance and vitality through insufficient food intake, may be an indirect though positive factor in the development of pulmonary tuberculosis.

DR. PATTON—Dr. Honsberger, have you anything to say in closing?

DR. HONSBERGER—I just want to take this opportunity to express to Dr. Ward and Dr. Van Dervoort my appreciation of the splendid contributions they made in this discussion.

DR. PATTON—The question of disability in connection with life insurance has become so much a part of our daily work that I felt

is was an appropriate time for us to consider some of the probable causative or underlying conditions that give rise to our claims. None of our members have given more time and attention to these questions than has Dr. Harold W. Dingman, who brings to us "Disability Insurance on Digestive Complainants." Dr. Dingman, who, we know, is very short and pertinent in his sentences, has said that about fifteen minutes will be sufficient. We find that we will have about that time before luncheon. We will have a discussion of his paper immediately following luncheon. Dr. Dingman.

DISABILITY AND DIGESTIVE DISORDERS

DR. HAROLD W. DINGMAN,

*Vice President and Medical Director
Continental Assurance Company.*

My valued friend, Allen Patton, asked me to discuss the subject of "Disability and Digestive Disorders" and I confessed to him then, as I must again now, that I know very little about it. He was gracious enough to profess equal ignorance and I am quite content to be of equal ignorance with Dr. Patton on this subject if only I could hope to be of equal knowledge on other subjects.

Statistical insurance material is not available, at least not to me, in sufficient degree to draw dependable conclusions. But our applicants wait not for the experience that they only can give us, and they present themselves to us, and we handle them, as indeed we must, on the basis of judgment. It is my belief that our judgment should be tempered with conservatism until we acquire an adequate experience. We must remember that insurance policyholders have demonstrated a disability rate that has increased steadily and remorselessly since the first table was built in 1824. We must hold in mind that latter day experience with non-cancellable accident and health policies, and life companies' disability contracts, has emphasized the need of caution. Our clients appear to have chosen their coverage more advantageously than our companies have chosen our customers.

Indigestion may be due to many things. Suffice it in this brief paper to consider two causes only, but those two very important, gall-bladder trouble and peptic ulcer. These two conditions are more closely associated than is generally understood. The lymphatic vessels of the gall-bladder run downward along the common bile duct to become anastomosed with other lymph vessels that drain from the head of the duodenum and the head of the pancreas. Infection in the duodenum may easily be conveyed to the gall-bladder, or vice versa. The nerve control of

these two tissues is similarly close, for the same vagus that acts as secretory nerve to the gall-bladder is also secretory to the stomach, and is also secretory to the pancreas. With this close interrelationship in lymphatics and nerve supply, it is not difficult to understand that the condition that is thought to be gall-bladder may actually be duodenal, or pancreatic, and if indeed it is gall-bladder in origin, it may also be duodenal, or pancreatic. Accordingly it is hardly surprising to observe with Smithies (1) that 1000 consecutive cases of operatively demonstrated gall-bladder disease showed peptic ulcer associated in 7 per cent, pancreatitis in 6.5 per cent. Appendix disease was present in the astonishingly high proportion of 68 per cent. It further illustrates the point when we note Schutte's (2) recent statement that complications were present in 262 of his series of 371 surgical gall-bladder cases. Conversely, Alvarez' (3) large experience revealed to him that definite biliary tract disease is present in 8 to 11 per cent of men, 13 to 27 per cent of women, who have peptic ulcer.

BILIARY TRACT DISEASE.

Surgical treatment of gall-bladder trouble appears to give satisfactory results in 7 out of 10 cases. Such was Davey's (4) experience when he made a questionnaire study of 144 cases: 70 per cent were considered cures, 22 per cent relative cures. So also Auschutz (5) as he traced the survivors of 712 surgical cases. Krogus (6), too, found 70 per cent free from symptoms when he followed up 103 persons who had been operated on between 1912 and 1920. Deavor and Bortz (7) reported 65 per cent to be relieved. Their experience embraced 872 cases. Marks (8) was able to learn about 68 surgical gall-bladder cases at least two years after operation and considered 61 per cent to have complete relief. Tanner (9) could concede only 55 per cent as cured in his 117 cases. Steden (10) was even more pessimistic. In a series of 164 cases he found but 34 per cent free from pain.

Seulberger (11) has believed that 8 out of 10 persons were free from symptoms when he traced 217 patients who had had operation for gall-stones between 1912 and 1920. But 80 per cent seems somewhat optimistic. Sir Berkeley Moynihan (12) has written that about 20 per cent of individuals operated on by him for gall-stones had had previous operations for similar trouble. Dahl-Iverson (13) found his recurrences practically the same, 19 per cent, when he re-examined 196 gall-bladder cases, 156 of them removal, 40 of them drainage.

All in all it seems reasonable to conclude that satisfactory results follow gall-bladder surgery in 7 of 10 cases. The immediate prognosis after operation is grave, the ultimate prognosis encouraging. Most surgeons prefer removing the gall-bladder rather than draining it. Yet Darner and Cullen (14) were able to report 7 in 10 without symptoms after cholecystostomy was done in 290 cases.

PEPTIC ULCER.

Peptic ulcer is a disease of unknown etiology, as Fremont-Smith and McIver (15) remarked. Its cause is complex, Balfour (16) commented, and the great variations in the manifestations and complications of the disease emphasize the incompleteness of knowledge of the subject. "I am in the dark at present," said Lewisohn (17), "as to the real cause of duodenal ulcer."

Infections appeared to be etiologic in 42 per cent of 200 cases of duodenal and gastric ulcers in the experience of Friedenwald and Morris on (18), arteriosclerosis in 26 per cent, anemia in 28 per cent. Nervous and endocrine disturbances were present in 22 per cent.

Smithies (19) searched for causative conditions in 522 histologically proved chronic gastric ulcers and infections seemed to be responsible in 33 per cent. He classified etiologic factors as follows:

	Cases	%
1. Infections	173	33.7
2. Arteriosclerosis	77	14.7
3. Visceral hypertonia	68	13.0
4. Chronic general anemia	61	11.3
5. Syphilis	41	7.8
6. Visceral hypotonia	27	5.2
7. Postoperative	27	5.2
8. Industrial intoxication	22	4.2
9. Metabolic dysfunction	18	3.4
10. Trauma	8	1.5
	<hr/> 522	<hr/> 100.0

Parenthetically it is worth comment that hyperchlorhydria has little if any relationship to peptic ulcer. Friedenwald and Morris observed it in 17 per cent of their 200 cases, Smithies in 30 per cent of his series. But Smithies found almost the same number of persons with acidity absent or scanty, and the symptom-complex gave no clue as to who was hyper and who hypo.

There is abundant reason to conclude as Smithies did, that we have no basis for regarding peptic ulcer as a distinct disease entity. It seems more proper to consider that ulceration of the gastric mucosa is a local accident in the course of a systemic upset, the nature of which may be extremely varied.

Little wonder, then, that treatment directed to the "local accident" gives us after-results that are far from conclusive. To effect a cure, remove the cause. But we do not know what the cause is. Wherefore it should not be surprising to us to find that end-results of treatment are somewhat discouraging. Only 30 per cent of 96 men remained well, 40 per cent of 118 women, when David Smith (20) traced 214 persons who had been medically treated at the Royal Infirmary of Glasgow 5 to 15 years previously. It must be that this group were too poor to take proper care of themselves. Yet White (20) found only 30 per cent of 54 gastric ulcer cases who were entirely well when he traced them 3 to 5 years later.

But most observers have thought they had better results. White himself did, with 152 duodenal cases: 57 per cent. Apparent cures were 58 per cent 2 to 10 years after Blackford and Bowers (22) treated 86 cases (80 duodenal, 6 gastric). Smithies

(23) claimed cessation of the ulcer process in the high proportion of 77 per cent of 470 cases 2 to 10 years after non-surgical treatment. (306 were duodenal, 128 gastric, 36 pyloro-duodenal.)

Terminology is so various that it is difficult to compare the follow-up conclusions of different observers. Gastro-enterologists could aid their studies if they adopted a nomenclature as understandable as that employed by specialists in tuberculosis. The word cure is used now and then. When is a cure in peptic ulcer? I do not know, but after nine medical cures, W. J. Mayo once remarked, the patient should look to surgery for relief. Whereupon the internist might almost be expected to retort—and then return for further medical treatment.

We like to believe with Moynihan (24) and Bevan (25) and Balfour (26) that gastro-enterostomy cures about 9 of 10 persons who have duodenal ulcer. When Starlinger (27) made a follow-up study of 250 surgical cases in the Vienna and Innsbruck Clinics, between 1902 and 1926, 9 out of 10 had satisfactory results.

But Conybeare (28) could find only 6 in 10 who were considered relieved when he traced 108 persons who had been surgically treated at Guy's Hospital three or more years previously. Lewisohn (17) believed that less than 5 in 10 were cured when he re-examined in 1923 some 92 patients who had had gastro-enterostomy performed between 1915 and 1920. Whereupon he discarded the gastro-enterostomy operation in favor of partial gastrectomy, and many there are who have, Hedlund (29), for instance, a Swedish surgeon. When he obtained after-histories of 485 peptic ulcer cases, gastro-enterostomy gave satisfactory results in only 3 out of 10, but resections brought about recovery in 6 and 7 out of 10. Okinczyc (30), too, found better results with resection. He traced 197 duodenal ulcer cases that were surgically treated and regarded his successes as 8 in 10 when resection was done, 2 in 3 with gastro-enterostomy.

It is puzzling to attempt to reconcile clinical views that differ so widely. Many, many observers could be cited, but why? The point is, that when unanimity of medical opinion is lacking, the insurance attitude must be one of caution.

It will be helpful indeed when the literature records more such prognostic studies as that of Fremont-Smith and McIver (15). They reviewed the records of 678 peptic ulcer cases treated surgically at the Massachusetts General Hospital during 1919-23. Gastro-enterostomy was the operation that was usually done. Late results were obtainable in 472 cases and 66 per cent of the duodenal cases were found to be symptom-free, 60 per cent of the gastric cases. Both duodenal and gastric cases showed severe recurrence of symptoms in about 11 per cent of cases within 2 years, 14 per cent in 4 years, 19 per cent in 6 years. Alvarez (3) drew attention to this same increasing tendency toward recurrence when he addressed the medical directors last spring. "For the first few years after operation, the results are generally good, but as years pass, an increasing number of the patients suffer relapses; from 5 to 10 per cent have to be operated on again, and many become more or less incapacitated." It has been the insurance attitude since Arthur Hunter's (31) study of Mayo Clinic material in 1919, that the hazard is a decreasing one. A further study of larger material may show us otherwise.

To discuss the prognosis of peptic ulcer, comment must be made of the cancer hazard. Perhaps no controversy in medical literature is more interesting than the relationship of cancer to stomach ulcer. It is generally agreed that cancer follows duodenal ulcer in substantially less than 1 per cent of cases. But there is no agreement as to the probable incidence following gastric ulcer. As far back as 1909 and 1910 W. C. MacCarty (32)(33) at the Mayo Clinic was believing that as high a proportion as 71 per cent of stomach cancer was ulcerous in origin, and 68 per cent of stomach ulcers were carcinomatous in character. John B. Deaver (34) and Sir Berkeley Moynihan (35) have expressed themselves as believing that the cancer hazard

with gastric ulcer is a very grave one. Yet G. W. Criles' (36) experience is a very large one and so is J. Ewing's (37) and they have written that they believe that the cancer incidence is only about 5 per cent. The same diversity of opinion exists in Germany as here. H. Finsterer (38) wrote a very interesting essay on the subject in 1925 in which he quotes Aschoff as believing that ulcer carcinomata were extremely rare while Zenker considers almost all carcinomata to be ulcerous in origin.

Again the thought becomes impressed upon us that if clinical opinion is so very much at variance, it may pay us well in insurance to remain conservative until we know more definitely whether applicants with ulcer history will give us long illnesses because of cancer.

PSYCHICAL CONSIDERATIONS.

Nor should we forget the mental attitude of the individual who has had serious intra-abdominal trouble. Not infrequently he becomes scared, possibly panic stricken. Oftentimes he gets intensely introspective and self-analytical, and over-stresses minor symptoms. Any listener who has had a serious surgical disablement will be sympathetic to what I am saying. Any listener who has an aunt or mother-in-law thus affected will be less sympathetic, possibly, but in entire agreement with me that upper abdominal surgery produces a profound psychic effect.

Thought carries on to the widely varying mental attitudes toward sickness that different persons have. Some individuals give up to the idea of sickness more quickly than others and some seem to be stubborn in resisting the idea of recovery. It is very difficult to make adequate appraisal of what might be called disability-capacity, but we should not disregard such obvious truths as these. We know so by general observation as well as insurance experience. Women have more sicknesses than man. Southerners are disabled oftener than Northerners. Slavs admit longer illnesses than Nordics.

The psychic element is potent in many persons, so potent that we may wish to agree with McLester (39) when he stated that

indigestion is a psychoneurotic affair in about one case in three. Of 5700 persons consulting him in Birmingham, Alabama, 28.5 per cent had digestive complaints chiefly, and 33 per cent of these were psychoneurotics, were evidently born so and were regarded as predestined to their troubles because of constitutional inferiority. As J. Ramsay Hunt (40) has reminded us, many disorders are purely psychic and many others result from psychic influences. The mind bears the burden of all disease of whatever the nature.

In connection with the psychic, we all of us know that we are getting disability claims thrust at us for conditions like angina pectoris, diabetes, chronic Bright's and high blood pressure, and if we hold in mind the wording of our disability clauses, we purport to pay for total and permanent disability. The point is this: The vast majority of these individuals are not totally disabled; they are partially disabled, if anything; and our clauses are not designed for prophylaxis; the coverage is not designed for the angina pectoris individual or for the diabetic individual who wants to give himself the best chance possible, proper as such an attitude is. I might have the same attitude. If I have some capacity left in me for working, then I am not totally disabled and then I am not entitled to coverage under the clauses that most of us are writing. I can't over-stress that point. Our claims are not for prophylaxis.

ETHICAL CONSIDERATIONS.

Nor dare we disregard the honesty of our clients. It has been my contention for some time that our applicants should qualify ethically, and psychically, as well as physically. It is imperative that we select policyholders who will be frank and honest when applying to us for indemnity. The disability problem would be solved if none but the unimpaired got policies and none but the surely impaired collected indemnities. In a brief survey of a certain portion of the non-cancellable health and accident business in our Company, drawing three broad groups according to ancestry, we found indemnity payments to vary as

Disability and Digestive Disorders 293

greatly as \$6.00 to \$3.00 to \$1.00 per unit of premium. Classifying these claimants according to cause of disability tells us little as compared to classifying them according to their personal and racial characteristics.

INSURANCE CONCLUSION.

Peptic ulcer and biliary tract disease should be regarded as surgical ailments. When known to be present, they are not acceptable disability risks before operation. After operation present knowledge makes it appear that further trouble may be expected in about one third of the gall-bladder cases, one third of the duodenal ulcer cases, perhaps one half of the gastric ulcer cases. Conservatism is advisable.

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DR. PATTON—Luncheon is ready. We will re-convene promptly at 2:30 and a discussion of Dr. Dingman's paper will be the first topic this afternoon.

AFTERNOON SESSION.

DR. PATTON—Those of us who have had our training in life insurance and then have had to meet, right off the bat so to speak, the disability claims, have had a difficult road to travel.

Feeling that a training in handling these cases for an office that has had life, accident, health and disability cases for their decisions, would bring valuable knowledge to us, we will now call upon Dr. McLeod C. Wilson of the Travelers.

DR. WILSON—Dr. Dingman in discussing the subject of disability and digestive disorders selected two outstanding ailments, namely, Ulcer and Disease of the Gall Bladder. These two conditions give rise to many Disability Claims. Dr. Dingman lists ten causes of ulcer or gall bladder disease, from the research work of Smithies. This list makes it seem probable that about thirty-three percent are dependent upon infections. I think it is generally conceded that focal infections play an important part in the production of digestive disorders. If it were possible to learn of existing foci of infection and the disability clause eliminated in these cases, the loss ratio for digestive disorders would undoubtedly present a very different picture. In a recent review of some Disability Claims I found a history of tonsillectomies in one-fifth of the cases of gall bladder disease, all having been performed within a few years preceding the onset of disability.

Discussion—Disability-Digestive Disorders 295

While without doubt infections are an important factor, we rarely get such in our histories unless there has been an attempt to remove same by surgical interference. Infected conditions of the tonsils, teeth, sinuses or other parts, unless they have given rise to definite disability are rarely brought out in an insurance examination. The individual evidently considers them unimportant and the examining physician usually considers such information too trivial to put into his report.

A hurried review of five hundred Disability Claims for disease of the digestive tract emphasizes the fact that important histories were conspicuous by their absence; many such histories, however, were brought out during the investigation of the Claim. This review, while it did not aid much in learning causations, nevertheless indicated the necessity of complete examinations in those having vague abdominal symptoms. Dr. Dingman speaks of the close association of the gall bladder, duodenum and pancreas. While it was impossible to get any definite figures on this condition, it was very evident that many of our gall bladder cases were complicated by stomach or pancreatic trouble or vice versa. It was of interest to learn that in seventy-seven cases of ulcer, the appendix had been removed within five years in sixteen percent. This would indicate that the appendix was blamed for symptoms when the actual pathology was located in the stomach or duodenum.

Dr. Dingman draws our attention to the fact that in gall bladder cases recurrence is a decreasing hazard, particularly in cases in which the gall bladder had been removed. In contradistinction to this there seems to be an increase in tendency toward recurrence in ulcer cases, at least during the first few years. This seems to me to be correct as the observation of a large number of Health Claims bears out this point, although I have made no statistical study of the subject. In the study of Life Disability Claims histories of gall bladder disease or ulcer have not entered into the picture as they were not granted the Disability Clause. There was one exception and that is, we have been accepting operative gall bladder cases where the gall bladder had been re-

moved, and the experience has justified such action. From the information gained by our own experience and that obtained from the various authorities, it is obvious that applicants giving a history of ulcer cannot be considered for this form of insurance, and gall bladder cases must be limited to those having had cholecystectomies.

In review of Disability Claims above referred to, it may be of interest to cite a few of the more prominent diseases of the digestive tract and their relative frequency. It was necessary to throw out a few cases so that the following figures are based upon 449 Claims. The mortality figures will seem excessively high, but probably not when we consider that all of these cases were more serious than the average, and had already produced a disability of three months. Recoveries refer to those cases which had again engaged in business and the Disability Benefits withdrawn. It is very probable that many of these cases listed under "recoveries" will again become disabled.

Carcinoma of Stomach.

45.2	Average Age
8.8	Mos. Average Duration
19%	Of Total
60%	Mortality
	No Recoveries
	Balance Still Running

Carcinoma of Pancreas.

48.5	Average Age
6.1	Mos. Average Duration
2.2%	Of Total
99%	Mortality
1	Recovery

Carcinoma of Oesophagus.

42	Average Age.
6.1	Mos. Average Duration
1.5%	Of Total
100%	Mortality

Tuberculosis of Intestines.

29.4	Average Age
14.3	Mos. Average Duration
2%	Of Total
65%	Mortality
2	Recoveries
	Balance Still Running

Ulcer of Stomach.

36	Average Age
6.7	Mos. Average Duration
7.5%	Of Total
25%	Mortality
65%	Recoveries
	Balance Still Running

Appendicitis.

33.5	Average Age
5.1	Mos. Average Duration
10%	Of Total
28%	Mortality
65%	Recoveries
	Balance Still Running

Carcinoma of Colon.

41.6	Average Age
8.2	Mos. Average Duration
8.5%	Of Total
100%	Mortality
	No Recoveries

Carcinoma of Liver.

44	Average Age
6.3	Mos. Average Duration
4.3%	Of Total
100%	Mortality

Discussion—Disability-Digestive Disorders 297

<i>Cirrhosis of Liver.</i>		<i>Ulcer of Duodenum.</i>	
46.6	Average Age	39.5	Average Age
6.5	Mos. Average Duration	12.4	Mos. Average Duration
2.5%	Of Total	9%	Of Total
90%	Mortality	19%	Mortality
1	Recovery	66%	Recoveries
	Balance Still Running		Balance Still Running
<i>Ulcerative Colitis.</i>		<i>Gall Bladder Disease</i>	
34.2	Average Age	41.4	Average Age
11.4	Mos. Average Duration	12.1	Mos. Average Duration
2.4%	Of Total	6.5%	Of Total
18%	Mortality	21%	Mortality
63%	Recoveries	60%	Recoveries
	Balance Still Running		Balance Still Running

The above groups cover the five year period ending July, 1928. The periods of disability for the entire group are comparatively short, namely, eight months, appendicitis giving the shortest period and tuberculosis of the intestines the longest.

The Carcinoma Group were peculiarly free of histories. Investigations of Claims showed that symptoms were usually noted about six months prior to the commencement of disability. When disability began the course was uniformly rapid. In connection with the Colon Group of Carcinomas 27% had had an appendectomy performed sometime in the past. In three of these cases the carcinoma developed in the caecum, the rest at various points in the colon. Why such a large number of appendix cases had appeared in this group, I do not know. Focal infections did not appear to be a point of interest in the ulcer or appendix group or, at least, were not mentioned in the histories. Dr. Dingman comments on the psychic effect of intra-abdominal trouble upon disability. I could not say that this was a material element in the duration of disability, however, it may be that upon a study of Claims for neurasthenia and allied conditions that we may find many that are dependent upon intra-abdominal troubles.

It is very evident that if we are to underwrite this form of insurance intelligently we must impress upon our examiners the need of careful histories, as it is only by the consideration of such that we can speculate upon the probability of future disease. The gross physical impairments that are brought out on examination

we can easily deal with, but if we are to weed out the undesirable disability risks by medical selection, we must have careful histories covering all departures from good health. Special comment should be required regarding the condition of the tonsils and teeth and the abdomen palpated for points of tenderness or masses.

No matter how careful we may be in our selection, the fact remains that the most important part in the underwriting of these risks is, first, the amounts issued including that already in force should be well within the earnings, second, the type of employment should have an element of permanency, third, the moral status and stability of the individual should be known, fourth, occupational hazards must be taken into consideration and certain vocations definitely eliminated. On the whole I do not find that occupation has any definite bearing on this group of diseases.

I might mention that our long term Claims of all character have been roughly investigated from a standpoint of occupation. This shows nothing conclusive, but does point toward longer Claims among certain professions than in the regular commercial lines. Also there seems to be a tendency for those whose incomes go on irrespective of their individual activity, to be longer than those who are solely dependent upon their individual endeavors.

DR. PATTON—A diversity of counsel many times brings one nearer to the solution of his problems. The next speaker had some of his earlier training in the same office as Dr. Wilson, but for a number of years has had to apply his knowledge and develop his opinions in different surroundings. We will now hear from Dr. Frank Harnden of the Midland Mutual Life Insurance Company.

DR. HARNDEN—Dr. Dingman is to be congratulated upon the capable handling of a difficult and somewhat obscure subject—obscure because of the haze surrounding indefinite opinions in many different quarters.

It is to be regretted that figures on this phase of underwriting are not available to light our way along this particularly dark road. If companies would set down on record their experiences equally in all lines, our task would be much less difficult.

Discussion—Disability-Digestive Disorders 299

Dr. Dingman has shown by a careful review of the literature available that the outlook following gall bladder surgery is not as gloomy as we are prone to believe. Particularly the surgery necessitated by cholelithiasis is curative in the great majority of cases.

Concerning gastric and duodenal ulcers, Balfour has to say in addition "that the normal tendency of chronic peptic ulcer is to recur. The intervals between recurrences may be long and usually in the remission the patient appears to be in perfect health, so that the repeated reappearance of the familiar symptoms becomes a disappointment to the patient, the therapist and the surgeon." We might very well add, in many cases disappointment descends upon the insurance companies to the extent of millions of dollars.

In cases in which we grant disability coverage and where the history, if complete, would include symptoms of digestive disturbance, again to quote Balfour, "it is fortunate that, as has been shown by experience and clinical evidence, attempts at healing are constantly taking place and many lesions heal without any treatment."

When we realize how often surgical treatment of these cases is at last resorted to when medical treatment has failed, disability protection is certainly poor underwriting when such a history is revealed. This is particularly so inasmuch as we know the disability of gastric ulcer is progressive.

In the consideration of cancer we have been told and experience shows that digestive disturbances are rare and late in time. However, with ulcer these symptoms occur early, are intermittent, and are nearly always related to the time of ingesting food.

Unfortunately for us as underwriters, the hypochondriac must be considered from time to time. Probably no applicant is more difficult to classify than the one who as a rule enjoys poor health but occasionally complains of feeling better. A clinician, even though capable and experienced, often cannot satisfactorily enlarge the personal horizon of one so afflicted.

Dr. Dingman mentions the honesty of our clients. At the risk of being abruptly ushered from the room, I cannot refrain from remarking that a disability habit, or as our essayist has cleverly

said, "disability-capacity", can be developed to completion in ninety days and from there on—what then?

One of the bright spots in a somewhat cloudy picture seems to be the statement made last year before this body by Dr. Olsen that a review of disability requests over a nine month period showed five claims due to gastrointestinal disturbances, one of which was sufficiently severe to be approved and four were disapproved.

I had hoped to give a word or two on the experience of my company with disability claimants who have had digestive disturbances in the past. Too many of our cases with such histories, however, have been inconsiderate enough to become disabled because of insanity or the result of arguing the right of way with a motor truck.

In my opinion Dr. Dingman scores a grand slam when he advises cautious and conservative underwriting of disability protection which we are including in life contracts.

DR. PATTON—It is pleasant to make friends in this life and to have them respond to your request when you feel the need of services you are sure they can render. Though the next speaker had his earlier experiences in the Home Office of a company that did not write disability insurance, yet for a number of years he has been in the thick of it. His company has had much to do with actuarial, medical and executive discussion of this feature of our business. We will hear from Dr. Lawrence G. Sykes of the Connecticut General.

DR. SYKES—I am in hearty accord with Dr. Dingman's statement that our judgment should be tempered with conservatism until we acquire an adequate experience. This is particularly true in our attitude toward Disability in ailments of the gastro-intestinal tract, especially peptic ulcer and conditions of the gall bladder; first because of the physical and second as a result of the psychical aspects. The latter has played an important role so far but not to be compared to that which it will in the future when Disability payments will make it possible to take advised protracted rest pe-

Discussion—Disability-Digestive Disorders 301

riods at our expense. Surgeons and internists are today advising prolonged rest periods as well as a let down in work where peptic ulcer is involved. Can anyone say it is not advisable or not indicated?

There is increasing evidence as a result of the Disability experience to date that Home Office Officials are looking to their Medical Officers for sound conservative underwriting of Disability. With the medical knowledge we have from the leading authorities in medicine and surgery just how can we justify our present system of rating peptic ulcers for life insurance, a rating of five, seven, ten or fifteen dollars a year per thousand of insurance for one to six years, covering the period when the policyholder is likely to be free from symptoms and not cured in many instances. The rating is then automatically removed without medical examination or X-ray study at a time when many leading authorities find that their patients are again coming under their observation for further treatment. We certainly are not justified automatically at the end of this rating period in recommending liberal Disability coverage when the source of the trouble is not removed and the etiological factor unknown as in peptic ulcer.

We are again indebted to Dr. Dingman for his emphasis on the psychical and the ethical considerations in this big question. Accident, Health and Non-Cancelable Disability Insurance have taught many things from which lessons can be learned and put into practice in underwriting Disability coverage in connection with Life Insurance. Let us remember the experience gained by companies in handling Health Insurance, that the question of rates is not going to solve the Disability problem in connection with Life Insurance for it was found in Health Insurance that with the increase in rates they had a higher loss ratio, the element of self-selection working against the companies. I therefore, again make a plea for sound, conservative underwriting of Disability even though the rates are raised.

DR. PATTON—Dr. Dingman, have you anything to add in closing?

DR. DINGMAN—Dr. Muhlberg has some splendid thoughts on one phase of the subject, and if he would favor us with two or three comments, I know they will be of interest to us.

DR. MUHLBERG—I think we must in the future take into consideration in acting on disability applicants and claimants, the tendency of modern medicine to regard a great many diseases as a cause for total and permanent disability, where formerly they were not so regarded at all. There is a tendency I think nowadays to consider a patient with a chronic peptic ulcer that does not yield to treatment or operation as being totally and permanently disabled; and so also there is a tendency of doctors to look upon advanced diabetes as being a cause of total and permanent disability.

There is good reason why they should look upon such diseases in this way, because if a man develops tuberculosis and the diagnosis is made in the incipient stages, we are perfectly willing to pay him total and permanent disability and still regard his chances of recovery as quite good. On the other hand, diabetes—at least the advanced form of diabetes—runs its course in from about seven to twelve years. I think Dr. Joslyn made the statement that under insulin treatment, a diabetic who formerly lived on the average about seven years will now live about twelve years. It is quite as logical therefore, to look upon a diabetic, who will in all probability die in twelve years in spite of the most careful treatment, as being just as totally and permanently disabled as a tubercular case, who in all probability will recover in four or five years.

I think doctors are beginning likewise to regard many forms of heart disease as being causes for total and permanent disability, even where such heart disease is not immediately disabling. I was very much struck, for instance, by what Dr. Henderson stated yesterday, that there is no doubt about the fact that in the human heart the ventricular extra systoles chasing themselves around the heart will cause a ventricular fibrillation which is promptly fatal. You will remember he stressed the point that many hearts that are not working well are likely to be thrown

Discussion—Disability-Digestive Disorders 303

into ventricular fibrillation simply because there is something wrong with the ignition. In other words, if a man has extra systoles of ventricular origin, that man really should be considered a chronic invalid, because at any time, due to undue excitement, business worries, etc., he is likely to throw his heart, on account of bad ignition, into a condition of ventricular fibrillation and die. In consequence, many doctors are beginning to take this viewpoint and are beginning to believe that recurring attacks of angina pectoris, auricular fibrillation, high blood pressure and other diseases calling for mental and physical repose, should be regarded as totally and permanently disabling the patient, particularly since mental ease and physical rest are part of the prescribed treatment and that death may be precipitated through anxiety, worry, emotional conditions or physical overwork.

I have in mind particularly, two disability claims in our own Company. Both happen to be physicians.

One doctor is a very good surgeon, but is of the neurotic type, undoubtedly. Under ordinary circumstances, his blood pressure is not abnormally high, running somewhere between 140 to 160. After a surgical operation, however, his blood pressure mounts considerably above 200. His physicians have advised him that he must no longer operate, because it will unquestionably permanently raise his blood pressure and he may even die suddenly in the operating room. He brought claim against us for disability, which I recommended be not paid. Unfortunately, we were weakened in our position because another company having a smaller policy promptly paid disability benefits to him. To our protests, he stated: "All right, if you don't pay me my disability, I will go to court. I have a dozen doctors who will appear in court and state that I am incapable of doing any work, particularly the work that I am fitted for."

The other physician is a diabetic and is on a very limited diet. He makes a claim, stating: "I can live on this limited diet, but as you know, if I do any work I will need more calories to keep the engine going. If I consume more calories, the sugar appears

in my urine. I am not going to consume any more calories than I absolutely must, because it will hasten the progress of the disease." We disputed that claim, but nevertheless, we are now paying it.

I believe we are going to have more of these cases in the future—I am afraid so, at least. Doctors are diagnosing heart disease and other chronic diseases with greater accuracy. Formerly, if a man had auricular fibrillation or chronic diabetes or high blood pressure, they sent him back to work and let him die in harness; but nowadays, physicians feel (and correctly so) that the lives of these patients can be prolonged by rest, freedom from mental and physical strain and in many cases, hospitalization.

Dr. Dingman made a very good point in stating that we ought to consider the psychic element as well as the element of moral hazard in the selection of disability applicants, and we ought likewise, show more unanimity in our action on these cases, not only in selecting the risks, but likewise in recommending the payment of the disability claims.

DR. PATTON—Dr. Dingman.

DR. DINGMAN—Mr. Chairman and Gentlemen: A paper of itself need not be good to be valuable if only it is provocative. Those who discussed the paper have made the subject good, and I am very thankful to Dr. Wilson and Dr. Harnden and Dr. Sykes and Dr. Muhlberg for the intelligent comments that have been made.

I am constrained to emphasize a point or two. Presumably, we are offering total and permanent disability protection, but as Dr. Muhlberg put it before I stood up—I am using his term—what we are paying for in many cases is partial and prolonged disability. It is not the intent of the companies to do so. I don't know a reason in the world why a man who may have had an attack of angina pectoris in 1928 should be disabled in 1929, nor do I know a reason in the world—speaking now from a company standpoint—why a man who may have an attack of angina pectoris in November, 1929, should expect to be disabled in

Discussion—Disability-Digestive Disorders 305

November, 1930. I quite grant you if I have angina pectoris I may wish not to work, and if my economic circumstances permitted, I may be well advised not to work, but that desire of mine does not constitute a total and permanent disability. It is total and permanent disability coverage that we are selling, not prophylaxis for disability.

Dr. Wilson made another comment in personal conversation that I can not forbear repeating. He spoke of a very well known surgeon who said: "I rather see how you can write life insurance, but I don't see how you can write health insurance. I have quite a few patients whom I advise to accept immediate operation and they say, 'Well, I must wait; I am going to get some disability protection'."

Consequently, as we discuss the disability of digestive disorders, it is pertinent to know how often these things may come and how long they last when they do come, and it is just as important to figure out if possibly they have not already come, the history being concealed to us. And that is ethical. Also we should anticipate if the individual will require for himself a twelve month disability where the average individual will take three months. And that is psychical, the mental attitude. So in this disability game there seem to be three distinct phases: The physical, the psychical and the ethical.

Gentlemen, I thank you.

DR. PATTON—A resolution was passed yesterday at our meeting relative to the appointment of a committee to draft a resolution in regard to Dr. Rogers. I desire to appoint on that committee, Dr. Willard, Dr. Weisse and Dr. C. T. Brown, our Secretary.

We have had many instances of the interest in the progress of our association that has been manifested by the representatives of the company with which the authors of the next paper are connected. They have in Dr. F. S. Weisse a worthy successor of medical directors who were immensely helpful in our formative years and the development of our organization and its later growth. Mr. Wendell M. Strong has previously con-

tributed in a joint paper to our proceedings and he adds for us today the actuarial viewpoint of an important group of insurance risks. Dr. Weisse, will you report the points of your paper for our consideration?

DR. WEISSE—I don't know that I have anything to say in addition to the paper. The principal point that was brought out was the fact that there has been a distinct improvement apparently in the female risks. Whether we have more carefully selected them, whether the women are better taken care of by the medical profession that they live longer, I don't know. It may be that they live more sheltered lives than men, that is, the women who apply for insurance.

One thing that we found that was interesting was the fact that apparently the larger amounts gave us a better mortality. Another very interesting thing that we found was that the lapse rate showing was less in women than in men. They held on to their policies longer. We found also a difference in the occupations among the women rather than the men. I think those were the three principal points that we brought out.

The policies, of course, on the women are not as large as on the men, and I don't know how the proportion of female lives runs in the other companies, but ours were about 9 per cent.

I am sorry to say that Mr. Strong has a bad cold and can't be with us today.

WOMEN AS LIFE INSURANCE RISKS

EXPERIENCE OF THE MUTUAL LIFE INSURANCE
COMPANY OF NEW YORK

WENDELL M. STRONG, *Associate Actuary*,

AND

DR. FANEUIL S. WEISSE, *Medical Director*.

The Mutual Life Insurance Company of New York has insured women since it began business in 1843. In fact the first death claim paid by the Company was under policy No. 332 for \$2,000, on the life of Jane Coit of Plainfield, Connecticut, who died on March 12th, 1844, of pulmonary tuberculosis, six months after issue.

From information obtained from the original register of the Company it would appear that beginning on or about June, 1855, we began charging an extra premium of $\frac{1}{2}$ of one per cent on the lives of women. This extra was charged between ages 16 and 48. These old records did not show that we charged the extra premium in every case between these ages and it may be that there were exceptions, the reason at this time not being known, only that certain policies did not show this extra charge. There may have been physical reasons.

In our search through the old records we find under policy No. 12001 issued June 29th, 1853, on the life of Annie M. Smith, age 24, a charge of 1% extra for the first year only, the applicant being pregnant, and no further extra is mentioned.

In the Eighteenth Annual Report of the Company, dated 1861, it states under details of rates that $\frac{1}{2}\%$ in addition was charged on the lives of women under age 48, and this would appear to apply to all women. This rule continued until December, 1875, when it was changed, the $\frac{1}{2}\%$ extra prem-

ium being charged until one year after the menopause. Under date of July, 1891, the rule was changed again making the extra premium payable to age 48 and under date of March, 1897, the Company discontinued the practice of charging extra premium on the lives of women.

In June, 1910, the Company cancelled the payment of existing extra premiums, i. e., the so-called "Female Extras" due thereafter on all outstanding policies on the lives of women.

Up to 1908 it was the practice to postpone acceptance of a married woman until two years had elapsed since the date of marriage or until 60 days after a normal confinement. In 1908 not only was this practice abandoned but we began to accept pregnant women—up to the seventh month—provided they had had a previous normal labor. Insurance on married women, as a rule, was not to be issued in favor of husbands nor on joint-life plan on husband and wife.

In 1866 women were considered less satisfactory risks than men as the following section of the Twenty-third Annual Report of the Company in that year shows.

"In addition to the above, if the applicant be a female, ascertain whether the functions of the uterine system are normal and regular; if miscarriages have occurred, state how often, under what circumstances, and how long since; whether confined since. If parturition have ensued, remark whether the labors have been easy and natural or difficult, and also whether puerperal fever or other disease has intervened. If the female applicant be between the ages of forty and fifty years of age, the greatest vigilance must be exercised in detecting any latent infirmity that may be likely to produce serious disease. Although it is probably true that women who, at fifty years of age, possess perfectly sound constitutions, have a greater prospect of longevity than men at the same period, yet the experience of all assurance companies reveals the fact, that for this country they have not proved profitable risks. This has been accounted for by the

difficulty of making thorough examinations, by the circumstance that most applications are made at that period of life when the dangers arising from parturition are greatest, and by the belief that the party in whose behalf the policy is issued often possesses knowledge of some weakness or hidden disease tending to the shortening of life. The Medical Examiner must therefore be on his guard in these applications, and should ascertain whether prolapsus uteri or leucorrhoea exist, or any symptoms denoting tumor or other diseases of the utero-generative system.

In cases of pregnancy, postpone the application until a sufficient time has elapsed, after the delivery, to indicate that the constitution of the party has not been affected thereby."

Table 1 gives a general comparison of the relative mortality rates among men and women for a period of thirty years up to 1915 and shows women as giving a better mortality than men—an improvement on 1866.

The next statistical study, Table 2, covers the issues of 1907 to 1915 exposed to 1916 and shows an improved mortality—the improvement seems greater among women than among men.

This study shows the mortality by different classes: Self-supporting spinsters, Class 1(a), shows an unusually low mortality both by policies and by amounts. Dependent Spinsters, Class 1(b), and Widows and Divorcees, Class 4, though not so good as 1(a), show mortalities better than the men. Married women—husband beneficiary—Class 2—and beneficiary other than husband—Class 3—taken together at ages under 45—give a mortality by policies of 101%—as compared with the mortality for men for all ages of 78%. In these same two groups taken together at ages 45 and over, the mortality by policies is 55% as compared with 78% of the men. This shows 23 points above the men for the younger group and 23 points below the men for the older group.

The percentage of both deaths and losses of the "Total Women—all Classes combined" are distinctly lower than

those of the men in all policy years and in all ages excepting age group 15-19. Although practically all in this age group were spinsters at date of issue a great number of them must have married later. The puerperal hazard was a large one. Examination of the causes of death shows 31 deaths from that cause in Classes 2 and 3 among the entrants under age 45. There were also 31 deaths from cancer among entrants under age 45—six times as many as among the men. These two causes of death are apparently responsible for the higher mortality among the women under age 45.

In 1925 we made another study of our female risks covering the issues of 1907 to 1922 exposed to 1924. This is presented in Table 3 which is divided into the same five classes as Table 2.

The proportion of policies issued to women had increased to 8.5% and to 5.3% of amounts. The mortality ratios were excellent for Class 1(a) and Class 4 throughout. Class 1(b) was very good for the first five years and about equal to the men's experience for years 6 and over. Classes 2 and 3 taken together were not good, especially at the younger ages—for entrants ages 15 to 29 the ratios for the first five policy years were 101% by policies and 97% by amounts; for policy years 6 and over, 110% by policies and 104% by amounts and for all policy years combined 104% by policies and 99% by amounts. Detailed data for this class are given because of its peculiarity. For all ages and all policy years combined the mortality of these groups was 4% higher than the men (83%-79%) by policies and 4% lower by amounts (79%-83%).

In Table 4 is given our experience, brought up to date. This is divided into issues of two periods, those of 1909 to 1918 and those of 1919 to 1927, and the two treated separately. This does not give the different classes of the previous experience but the results for women as a whole. The first page of this table, Page No. 321, contains a summary of the results of this experience.

Strong-Weisse—Women as Insurance Risks 311

Table 5 gives the relative mortality experience of 17 occupational classes, issues of the United States and Canada, for the years 1907 to 1924 inclusive, the observations carried to the policy anniversaries in 1925.

In addition to the entrants, exposures and ratios by the two standard tables for each occupation class, the experience of each class is also given.

The mortality experience of the seventeen occupation classes combined, which comprises about 92% of the total policies and 90% of the total amount insured to women in the same period, was considerably better than the general experience at each age period; furthermore, the rate of termination otherwise than by death of the same classes is also lower than the general experience.

We shall call attention to the occupation classes in which either the actual deaths or losses exceeded the expected by the M. A. Select Table in any of the three age groups.

Class 1, Actresses, shows very well for those insured to ages under 30. Above that age both the rate of termination and the mortality by policies is unfavorable.

Class 2, Chiropractors and Osteopaths, shows a favorable mortality for those insured under age 45, but unfavorably by policies for ages 45 and over. We also note that the rate of termination is higher.

Class 5, Factory Workers, shows a high mortality both by policies and amounts for those insured under age 30, but favorably above that age.

Class 7, Proprietresses of Hotels, Boarding Houses, Restaurants and Clubs, shows high mortality ratios for ages under 45, but normal for ages 45 and over.

Class 8, Housewives, shows high mortality for those insured under age 30, but normal above that age.

Class 9, Merchants, shows high mortality by amounts for the young entrants under 30, but normal above that age.

Class 12, Printers, shows excessive mortalities both by policies and amounts in each of the three age groups.

Class 14, Servants, Domestic and Private Families, shows likewise excessive mortality ratios both by policies and amounts in each of the three age periods.

Class 17, Telephone and Telegraph Operators, shows high mortality for those insured under age 30, but normal for ages 30 and over.

We have made a study of the death claims of issues of 1907 to 1922, exposed to 1924, with reference to causes of death (See Table 6).

The death rate was greater among men than among women for the following causes:

Typhoid Fever

Epidemic Influenza

Pneumonia

Main Degenerative Diseases—Apoplexy, Paralysis, Paresis, Heart Disease and Bright's Disease.

External Causes—Suicide, Homicide and Accident.

The death rate was greater among women than men from cancer. The comparison is not wholly satisfactory particularly with regard to the degenerative diseases because the average age at issue is nearly two years younger for women than for men.

There were 168 deaths from the diseases and conditions incidental to childbirth for ages at issue under 45. These deaths when related to the 407,884 years of life exposed at ages 15 to 44 correspond to a death rate of 41 per 100,000. Of these deaths, 75 occurred among those spinsters who married subsequently to the date of taking out insurance. These 75 deaths correspond to a death rate of 33 per 100,000 total spinsters at these ages. Eight deaths occurred among those widows and divorcees who subsequently remarried, which corresponds to a death rate of 20 per 100,000 of this class. The remaining 85 deaths occurred among married women, Classes 2 and 3. These deaths correspond to a death rate of 61 per 100,000 married women at ages at issue under 45 years. Of these 85 deaths 69 had had a normal pregnancy

Strong-Weisse—Women as Insurance Risks 313

prior to issuance. Of the 16 deaths which did not have a normal pregnancy prior to issuance, 3 died in the first policy year, 5 in the second policy year and 8 more than two years after the date of taking out insurance.

Taken as a whole, our women policy holders are better risks than the men and this holds true for most of the different groups.

(a) Their mortality is more favorable than that of the men.

(b) Unlike that of the men, their mortality is more favorable by amounts than by policies, indicating apparently that the larger amounts give the more favorable mortality.

(c) They are probably more persistent than the men—the proportion of lapses among them being 27% both by policies and by amounts as compared with the 33% by policies and 31% by amounts of our general experience. Further study by individual years might, however, show a different result.

The average policy on a woman is about one-half as large as on a man and the average age at issue of the women is 1.8 years younger than the average of the general experience. The proportion of policies issued on women's lives is about 9% of the Company's business.

TABLES.

In the following tables the data covered all policies issued to women in our agencies in the United States and Canada.

In Tables 2 and 3 the following classes are investigated separately:

Class 1(a) Spinsters, self supporting.

Class 1(b) Spinsters, dependents.

Class 2 Married women, husband beneficiary.

Class 3 Married women, any other beneficiary than husband.

Class 4 Widows and divorced.

For each investigation the numbers and amounts insured on women, and the percentages of the total insurance in the same field, were as follows:

Issues	Number	%	Amount	%
1907 to 1915	25,881	7.2	\$ 42,935,700	4.5
1907 to 1922	87,841	8.5	153,202,000	5.3
1909 to 1918	39,387	7.0	62,522,200	4.5
1919 to 1927	143,934	14.0	287,090,400	8.0
1909 to 1927	183,321	11.5	349,612,600	7.1

The expected is taken on the basis of the A. M. Select Table. The exposures ceased when the policy either became a paidup for a smaller amount or extended term insurance.

TABLE 1.
Issues of 1885-1906, Exposed to 1915.
Summary All Ages—All Policy Years Combined.

	Exposures		Actual		Approximate Ratio of Actual to Expected Deaths	
	No. Applications	Amount	Deaths	Losses	No.	Amt.
Men	6,716,196	\$18,426,364,900	66,305	\$197,440,600	100.2	106.7
Women	562,457	971,984,800	4,484	9,338,700	92.7	95.8

TABLE 2.
United States & Canada—Issues of 1907 to 1915, Exposed to 1916.
Summary by Policy Years, All Ages Combined.
Policy Year One.

Classes	Exposures		Actual		Ratio of Actual to Expected	
	No. Applications	Amount	Deaths	Losses	No.	Amt.
Total U. S. & Canada..	357,110	\$ 960,941,325	925	\$ 3,399,200	60.0	84.0
Total Men	331,229	908,006,625	895	3,309,000	69.2	85.3
Total Women	25,881	42,935,700	60	90,200	65.4	53.9
1(a)	11,023	14,214,500	10	10,500	28.3	22.3
1(b)	1,860	2,993,100	4	7,800	73.5	87.4
2	1,493	2,553,100	6	5,000	114.3	55.6
3	8,189	14,732,100	32	52,400	107.5	92.1
2 & 3	9,682	17,285,200	38	57,400	108.5	87.1
4	3,316	8,442,900	8	14,500	50.0	31.9

Policy Years One-Five.

Total U. S. & Canada..	1,071,376	2,872,301,225	4,255	14,436,700	76.8	83.4
Total Men	990,640	2,738,563,925	3,990	13,934,800	77.5	84.0
Total Women	80,736	133,737,300	265	501,900	67.6	68.9
1(a)	34,091	43,089,200	73	84,200	50.0	44.1
1(b)	5,889	9,244,600	15	22,500	66.2	62.1
2	5,230	9,075,900	19	33,500	74.6	76.6
3	25,192	45,389,000	117	198,400	92.7	81.5
2 & 3	30,422	54,464,900	136	231,900	89.6	80.8
4	10,334	26,938,600	41	163,300	56.0	76.3

Strong-Weisse—Women as Insurance Risks 315

TABLE 2—Issues of 1907 to 1915 (Continued).
Policy Years Six-Nine.

Total U. S. & Canada..	156,077	398,149,600	1,046	3,642,300	80.6	89.1
Total Men	143,507	378,467,400	976	3,516,300	81.3	89.7
Total Women	12,570	19,682,200	70	125,900	72.7	74.6
1(a)	4,928	5,802,300	19	18,000	63.4	49.0
1(b)	859	1,225,000	4	8,500	83.0	127.6
2	1,509	2,364,500	9	21,900	78.8	131.2
3	3,496	6,555,700	23	40,000	82.1	70.3
2 & 3	5,005	8,620,200	32	61,900	81.1	84.1
4	1,778	4,034,100	15	37,500	67.9	72.5

Policy Years One-Nine.

Total U. S. & Canada..	1,227,453	3,270,450,825	5,301	18,078,900	77.6	84.5
Total Men	1,134,147	3,117,031,325	4,966	17,461,100	78.2	85.1
Total Women	93,306	153,419,500	335	627,800	68.6	70.0
1(a)	39,019	48,801,500	92	102,200	52.8	44.9
1(b)	6,748	10,470,200	19	51,000	69.1	72.3
2	6,739	11,340,400	22	55,400	75.9	91.7
3	28,688	51,744,700	140	238,400	90.7	79.4
2 & 3	35,427	63,085,100	168	293,800	87.9	81.4
4	12,112	30,972,700	56	200,900	58.8	75.6

Summary by Ages, All Policy Years Combined.
Total Women—All Classes Combined.

Ages at Entry	Exposures		Actual		Ratio of Actual to Expected	
	No. Applications	Amount	Deaths	Losses	No.	Amt.
15-19	5,236	\$ 7,017,700	29	\$ 23,700	113.1	99.6
20-29	36,499	47,452,300	97	113,200	71.1	63.9
30-39	30,777	51,218,800	89	140,600	66.6	61.8
40-49	16,032	35,254,800	39	217,000	75.0	83.6
50-59	4,296	10,945,000	32	83,500	49.5	52.1
60 & Ov.	446	1,531,000	8	49,800	52.7	101.9
15-44	82,168	125,683,900	258	393,300	73.5	70.9
45 & Ov.	11,138	27,735,700	77	234,500	56.1	68.4
Total	93,306	153,419,500	335	627,800	68.6	70.0

Class 1(a)—Self Supporting Spinsters.

15-19	1,770	1,501,800	7	7,000	118.4	115.6
20-29	21,183	24,218,800	39	40,000	49.4	44.3
30-39	11,579	15,247,400	23	26,000	45.4	38.9
40-49	3,630	6,154,500	17	22,400	64.0	51.7
50-59	774	1,441,500	5	6,000	44.2	29.4
60 & Ov.	33	27,500	1	800	101.3	99.6
15-44	36,918	45,244,000	79	86,000	52.5	45.8
45 & Ov.	2,101	3,646,900	13	16,200	54.6	40.7
Total	39,019	48,801,500	92	102,200	52.8	44.9

TABLE 2—Issues of 1907 to 1915 (Continued).

Class 1(b)—Dependent Spinsters.

15-19 -----	3,127	4,797,960	12	16,200	113.0	99.3
20-29 -----	2,413	3,723,900	4	5,000	44.2	36.2
30-39 -----	714	1,056,600	--	---	---	---
40-49 -----	332	755,900	3	9,800	99.1	168.2
50-59 -----	101	140,500	--	---	---	---
60 & Ov. -----	1	1,000	--	---	---	---
15-44 -----	6,475	9,836,200	17	25,000	69.9	68.4
45 & Ov. -----	273	634,000	2	6,000	63.0	95.2
Total -----	6,748	10,470,200	19	31,000	69.1	73.3

Summary by Ages, All Policy Years Combined.

Class 2—Married Women, Husband Beneficiary.

Ages at Entry	Exposures		Actual		Ratio of Actual to Expected	
	No. Applications	Amount	Deaths	Losses	No.	Amt.
15-19 -----	67	\$ 88,500	--	\$ ---	---	---
20-29 -----	2,141	3,623,800	8	6,800	96.5	48.9
30-39 -----	2,817	4,815,500	12	26,100	92.7	117.4
40-49 -----	1,409	2,524,600	8	22,500	67.2	115.3
50-59 -----	214	287,000	--	---	---	---
60 & Ov. -----	1	1,000	--	---	---	---
15-44 -----	5,965	10,162,900	23	36,300	83.1	78.3
45 & Ov. -----	774	1,177,500	5	18,500	54.3	139.0
Total -----	6,739	11,340,400	28	55,400	75.9	91.7

Class 3—Married Women, Other Beneficiary Than Husband.

15-19 -----	265	322,500	1	500	111.7	46.0
20-29 -----	9,383	13,515,200	43	58,400	123.1	116.1
30-39 -----	11,530	21,093,400	43	70,500	85.3	76.3
40-49 -----	6,060	13,462,700	41	71,500	92.8	72.3
50-59 -----	1,364	2,804,400	10	21,500	49.8	52.0
60 & Ov. -----	106	546,500	2	16,000	62.3	98.3
15-44 -----	24,802	42,841,600	115	181,900	105.6	93.7
45 & Ov. -----	3,886	8,903,100	25	56,500	55.1	53.2
Total -----	28,688	51,744,700	140	238,400	90.7	79.4

Classes 2 & 3 Combined—Married Women.

15-19 -----	332	411,000	1	500	88.7	35.9
20-29 -----	11,504	17,139,000	51	65,200	119.0	101.6
30-39 -----	14,347	25,908,900	55	96,600	86.8	84.2
40-49 -----	7,559	15,987,300	49	94,000	87.4	79.5
50-59 -----	1,578	3,061,400	10	21,500	42.4	46.9
60 & Ov. -----	107	547,500	2	16,000	51.9	98.1
15-44 -----	30,767	53,004,500	138	218,500	101.0	90.7
45 & Ov. -----	4,660	10,080,600	30	75,000	55.0	62.7
Total -----	35,427	63,085,100	168	293,500	87.9	81.4

Strong-Weisse—Women as Insurance Risks 317

TABLE 2—Issues of 1907 to 1915 (Continued)

Class 4—Widows and Divorced.

15-19	7	7,000	—	—	—	—
20-29	1,399	2,370,500	3	3,000	56.9	33.5
30-39	4,137	9,006,900	11	18,000	59.7	43.8
40-49	4,421	12,362,700	20	90,800	60.5	98.4
50-59	1,843	6,271,600	17	56,000	60.4	61.0
60 & Ov.	305	955,000	5	33,000	48.5	104.0
15-44	8,006	17,598,500	24	63,500	60.9	71.5
45 & Ov.	4,104	13,374,200	32	187,300	57.3	77.6
Total	12,112	30,972,700	56	200,800	58.8	75.6

TABLE 3.

United States & Canada—Issues of 1907 to 1922, Exposed to 1924.
Summary by Policy Years, All Ages Combined.
Policy Year One.

Classes	Exposures		Actual		Ratio of Actual to Expected	
	Number Policies	Amount	Deaths	Losses	No.	Amt.
Total U. S. & Canada	1,027,774	\$2,896,931,500	3,311	\$11,139,300	83.9	90.5
Total Men	939,933	2,743,729,500	3,087	10,752,300	84.9	91.7
Total Women	87,841	153,202,000	224	387,000	71.9	66.4
1(a)	40,284	56,825,800	71	83,100	55.2	44.6
1(b)	5,494	9,197,000	10	13,300	62.9	49.7
2 & 3	30,932	60,510,400	109	223,900	95.8	96.5
4	11,131	26,678,800	34	66,700	63.9	48.6

Policy Years One-Five.

Total U. S. & Canada	3,752,587	10,425,577,200	16,961	54,653,400	82.9	85.2
Total Men	3,436,343	9,886,801,300	15,825	52,702,700	83.5	86.1
Total Women	316,244	541,775,900	1,156	1,955,700	74.9	67.7
1(a)	146,161	199,936,200	402	509,800	64.3	53.0
1(b)	20,454	33,689,900	53	80,500	73.8	62.6
2 & 3	109,478	212,550,700	506	935,400	91.1	81.7
4	40,151	95,599,100	190	430,000	66.6	56.2

Policy Years Six-Ten.

Total U. S. & Canada	1,506,291	3,529,828,000	10,737	30,884,400	81.8	84.4
Total Men	1,366,843	3,365,180,400	10,110	29,793,900	82.4	84.8
Total Women	109,448	164,647,600	627	1,090,500	73.4	73.9
1(a)	48,634	57,979,200	229	275,900	75.5	72.9
1(b)	7,849	11,410,100	33	42,900	80.2	72.7
2 & 3	38,902	64,926,300	241	476,700	75.1	81.9
4	14,063	30,332,000	124	295,000	65.8	64.8

TABLE 3—Issues of 1907 to 1922 (Continued).

Policy Years Eleven & Over.

Total U. S. & Canada...	502,318	11,116,360,600	5,001	15,623,800	84.1	96.7
Total Men	405,905	1,064,790,100	4,678	15,049,000	84.2	95.9
Total Women	36,413	51,570,500	323	574,800	81.6	89.7
1(a)	14,660	16,606,400	81	90,000	69.4	65.0
1(b)	2,570	3,550,100	18	35,400	109.5	161.4
2 & 3	14,155	21,568,700	155	240,200	95.0	90.8
4	5,028	9,851,300	69	209,200	69.3	97.0

Policy Years Six and Over.

Classes	Exposures		Actual		Ratio of Actual to Expected	
	Number Policies	Amount	Deaths	Losses	No.	Amt.
Total U. S. & Canada...	2,007,609	\$ 4,646,188,600	15,738	\$ 46,508,200	82.5	87.9
Total Men	1,861,748	4,429,970,500	14,788	44,842,900	83.0	88.3
Total Women	145,861	216,218,100	950	1,665,300	76.0	78.7
1(a)	63,294	74,585,600	310	365,900	73.8	70.8
1(b)	10,419	14,960,200	51	78,300	88.5	96.7
2 & 3	53,057	86,489,000	396	716,900	81.8	84.7
4	19,091	40,183,300	193	504,200	67.0	75.2

All Policy Years Combined.

Total U. S. & Canada...	5,760,196	15,074,765,800	32,719	101,166,600	82.7	86.4
Total Men	5,298,001	14,316,771,800	30,613	97,545,600	83.2	87.1
Total Women	462,195	757,994,000	2,106	3,621,000	75.4	72.3
1(a)	209,455	274,521,800	712	875,700	68.1	62.7
1(b)	30,873	48,650,100	109	158,800	80.1	75.8
2 & 3	162,585	299,039,700	902	1,662,300	86.8	83.0
4	59,242	135,782,400	383	934,200	66.8	66.3

Summary by Ages, All Policy Years Combined.
Total Women—All Classes Combined.

Ages at Entry	Exposures		Actual		Ratio of Actual to Expected	
	Number Policies	Amount	Deaths	Losses	No.	Amt.
15-19	29,899	\$ 40,959,500	87	\$ 112,200	81.0	76.5
20-29	188,511	254,966,700	656	817,300	87.2	81.0
30-39	144,491	244,240,900	588	882,000	79.3	72.0
40-49	75,887	159,841,600	477	1,129,400	67.9	78.8
50-59	21,337	52,269,000	244	527,000	59.3	54.7
60 & Ov.	1,980	5,716,300	65	152,500	68.4	67.1
15-44	407,884	633,227,600	1,570	2,311,200	80.3	74.8
45 & Ov.	54,221	124,766,400	536	1,309,800	64.0	68.8
Total	462,105	757,994,000	2,106	3,621,000	75.4	72.3

Strong-Weisse—Women as Insurance Risks 319

TABLE 3—Issues of 1907 to 1922 (Continued).

Class 1(a)—Self Supporting Spinsters.

15-19	12,384	14,117,900	33	85,000	75.7	70.8
20-29	117,349	143,722,900	352	398,800	75.6	70.6
30-39	55,983	77,737,200	186	248,700	65.3	64.4
40-49	19,486	31,925,300	105	146,400	60.9	54.1
50-59	4,067	6,722,500	34	45,500	47.3	39.9
60 & Ov.	186	296,000	2	1,300	28.5	12.7
15-44	198,163	255,837,000	633	769,300	71.0	66.8
45 & Ov.	11,292	18,684,800	79	106,400	51.3	43.7
Total	209,455	274,521,800	712	875,700	68.1	62.7

Class 1(b)—Dependent Spinsters.

15-19	15,847	24,667,500	48	66,700	82.8	74.5
20-29	10,613	17,266,500	32	37,000	75.2	53.8
30-39	2,661	4,082,100	19	36,300	135.0	171.3
40-49	1,349	2,039,900	9	17,800	66.6	92.7
50-59	387	678,100	1	1,000	13.3	9.7
60 & Ov.	16	16,000	—	—	—	—
15-44	29,850	46,978,500	105	148,800	86.9	79.4
45 & Ov.	1,023	1,671,600	4	10,000	26.1	45.0
Total	30,873	48,650,100	109	158,800	80.1	75.8

Classes 2 & 3 Combined—Married Women Summary by Ages, Policy Years One-Five.

Ages at Entry	Exposures		Actual		Ratio of Actual to Expected	
	Number Policies	Amount	Deaths	Losses	No.	Amt.
15-19	1,224	\$ 1,562,600	5	\$ 10,000	126.3	194.1
20-29	36,783	58,465,000	160	241,200	117.9	111.8
30-39	42,965	84,679,100	178	277,000	98.6	77.8
40-49	23,017	53,699,600	120	318,700	76.2	87.5
50-59	5,182	13,111,200	42	87,500	60.5	49.7
60 & Ov.	307	1,008,200	1	1,000	12.4	3.7
Total	109,478	212,550,700	506	935,400	91.1	81.7

Policy Years Six-Ten.

15-19	283	372,000	1	500	82.6	31.2
20-29	12,077	16,964,400	54	77,800	97.5	99.3
30-39	15,883	26,455,400	67	96,200	66.8	57.3
40-49	8,674	17,024,400	75	176,500	69.1	82.4
50-59	1,871	3,638,600	36	77,200	72.5	80.7
60 & Ov.	114	471,500	8	49,000	136.5	194.0
Total	38,902	64,926,300	241	476,700	75.1	81.9

Policy Years Eleven and Over.

15-19	101	111,000	—	—	—	—
20-29	4,259	5,737,500	38	44,000	169.9	145.7
30-39	6,030	8,881,500	51	74,800	96.6	94.1
40-49	3,033	5,625,300	42	87,000	72.4	81.5
50-59	680	1,006,400	23	33,900	84.3	83.3
60 & Ov.	32	99,000	1	1,000	42.2	13.5
Total	14,155	21,562,700	155	240,200	95.0	90.8

TABLE 3—Issues of 1907 to 1922 (Continued).

Policy Years Six and Over.

15-19 -----	384	483,000	1	500	60.2	23.9
20-29 -----	16,336	22,701,900	92	121,300	113.3	112.3
30-39 -----	21,913	35,436,900	118	170,500	77.1	69.1
40-49 -----	11,727	22,652,700	117	263,500	70.2	82.1
50-59 -----	2,551	4,644,000	59	111,100	76.7	81.5
60 & Ov. -----	146	570,500	9	50,000	109.4	153.0
Total -----	53,067	85,489,000	396	716,900	81.8	84.7

All Policy Years Combined.

15-19 -----	1,608	2,075,600	6	10,500	106.8	144.9
20-29 -----	53,119	81,166,900	232	362,500	118.0	111.9
30-39 -----	64,908	130,116,000	296	447,500	88.7	74.2
40-49 -----	34,744	76,352,300	237	582,200	73.1	85.0
50-59 -----	7,703	17,755,200	101	198,600	69.0	63.6
60 & Ov. -----	453	1,573,700	10	51,000	61.3	85.3
15-44 -----	140,449	249,246,900	674	1,098,400	93.9	85.6
45 & Ov. -----	22,086	49,792,800	228	553,900	70.8	78.2
Total -----	162,535	299,039,700	902	1,652,300	86.8	83.0

Summary by Ages, All Policy Years Combined.

Class 4—Widows and Divorced.

Ages at Entry	Exposures		Actual		Ratio of Actual to Expected	
	Number Policies	Amount	Deaths	Losses	No.	Amt.
15-19 -----	60	\$ 98,500	--	\$ -----	---	---
20-29 -----	7,430	12,810,400	19	19,000	63.7	37.3
30-39 -----	20,939	42,305,600	87	149,500	80.3	69.5
40-49 -----	20,308	49,524,100	126	383,000	65.5	83.6
50-59 -----	9,180	27,213,200	108	282,500	58.2	53.5
60 & Ov. -----	1,325	3,830,600	43	100,200	76.1	64.0
15-44 -----	39,422	81,165,200	158	294,700	69.8	63.1
45 & Ov. -----	19,890	54,617,200	225	639,500	64.9	67.9
Total -----	59,242	135,782,400	383	934,200	66.8	66.3

Issues of 1907 to 1922, Exposed to 1924.

Ratio of Actual to Expected by the A. M. Select Table.

Policy Years One-Seventeen.

Ages at Issue	On Policies			On Amounts		
	Men %	Women %	Difference	Men %	Women %	Difference
15-29 -----	93	86	7	102	81	21
30-44 -----	83	76	7	89	71	18
45 & Over. -----	73	64	14	81	68	13
Total -----	83	75	8	87	72	15

Strong-Weisse—Women as Insurance Risks 321

TABLE 4.

Issues of 1909 to 1927, Exposed to 1928.
Summary by Policy Years, All Ages Combined.

Policy Years	Exposed to Anniversaries in 1928											
	Issues of 1909 to 1918				Issues of 1919 to 1927				Issues of 1909 to 1927			
	Men & Women		Women		Men & Women		Women		Men & Women		Women	
	No. %	Amt. %	No. %	Amt. %	No. %	Amt. %	No. %	Amt. %	No. %	Amt. %	No. %	Amt. %
1	90.8	95.6	71.7	63.5	66.0	51.6	58.4	74.7	74.1	55.9	59.4	
2	92.9	90.0	69.0	66.7	65.2	70.3	58.8	48.8	75.6	76.2	61.3	52.4
3	89.8	88.6	86.0	78.7	65.5	75.3	53.7	60.2	75.5	79.7	62.9	64.6
4	88.1	92.8	88.2	92.5	65.8	69.9	62.7	55.4	75.9	78.6	71.2	65.7
5	87.3	83.2	84.6	86.9	66.6	69.1	53.8	55.6	77.1	75.1	65.8	65.9
1-5	89.7	89.7	80.4	78.6	65.8	70.2	56.0	55.5	75.7	76.8	63.0	61.1
6	82.6	91.1	81.0	76.3	60.3	67.9	47.6	53.3	73.0	79.4	63.3	62.9
7	74.3	76.3	66.2	64.6	69.1	70.7	47.3	36.7	72.4	74.0	56.0	51.4
8	77.9	77.4	66.6	62.5	67.7	69.1	38.4	31.2	75.1	74.6	57.6	51.3
9	75.9	77.8	58.9	62.8	76.2	75.1	59.1	51.2	75.9	77.3	58.9	60.4
10	78.9	78.8	64.2	56.7	-----	-----	-----	-----	78.9	78.8	64.2	56.7
6-10	77.9	80.2	67.2	64.4	-----	-----	-----	-----	74.7	76.8	60.4	56.8
11	73.5	81.3	63.5	72.7	-----	-----	-----	-----	73.5	81.3	63.5	72.7
12	79.0	90.2	64.6	85.9	-----	-----	-----	-----	79.0	90.2	64.6	85.9
13	81.8	89.8	83.7	73.4	-----	-----	-----	-----	81.8	89.8	83.7	73.4
14	79.3	81.0	82.7	79.3	-----	-----	-----	-----	79.3	81.0	82.7	79.3
15	86.6	94.8	78.6	73.8	-----	-----	-----	-----	86.6	94.8	78.6	73.8
16	77.4	83.2	57.6	49.2	-----	-----	-----	-----	77.4	83.2	57.6	49.2
17	76.7	80.5	42.0	26.6	-----	-----	-----	-----	76.7	80.5	42.0	26.6
18	77.7	87.0	66.1	80.3	-----	-----	-----	-----	77.7	87.0	66.1	80.3
19	67.4	71.3	39.1	32.1	-----	-----	-----	-----	67.4	71.3	39.1	32.1
11 & ov.	78.6	85.9	68.9	71.1	-----	-----	-----	-----	78.6	85.9	68.9	71.1
6 & ov.	78.2	82.8	68.0	67.4	66.1	69.7	46.8	44.3	76.3	80.1	63.5	61.0
Total	81.7	84.9	71.9	70.9	65.9	70.1	54.2	53.4	76.0	78.4	63.2	61.3

Ages at Issue	Summary by Ages, All Policy Years Combined.											
10-14	-----	-----	-----	-----	36.2	42.6	36.9	62.3	36.2	42.6	36.9	62.3
15-19	87.7	86.2	75.8	67.0	60.4	71.6	41.2	38.0	75.5	79.0	54.1	48.2
20-29	89.7	96.3	85.5	83.1	55.8	55.7	56.3	55.8	77.2	77.9	69.4	66.5
30-39	84.3	92.7	76.8	72.3	64.6	66.7	61.8	53.9	77.4	81.1	69.9	61.7
40-49	77.6	88.8	64.1	76.7	71.4	77.1	55.3	60.2	75.5	81.0	60.3	63.5
50-59	77.0	77.1	57.7	52.5	70.4	72.6	43.3	50.7	74.7	75.2	51.1	51.6
60 & ov.	78.0	76.2	58.0	66.4	70.4	66.5	40.3	34.2	75.1	71.7	50.0	47.5
Total	81.7	84.9	71.9	70.9	65.9	70.1	54.2	53.4	76.0	78.4	63.2	61.3

TABLE 4 (Continued).

Issues of 1909 to 1918 Inclusive.

Exposed from 1909 to 1928

Women Only.

Summary by Policy Years, All Ages Combined.

Policy Years	Exposed to Risk		Died		Ratio of Actual to Expected	
	Number	Amount	Number	Amount	No. %	Amt. %
1	30,387	\$ 62,522,200	101	\$ 153,500	71.7	63.5
2	34,689	55,355,200	118	199,100	69.0	66.7
3	33,563	53,737,400	153	251,200	86.0	78.7
4	31,290	49,479,600	159	294,700	88.2	92.5
5	29,567	46,233,800	158	287,000	84.6	86.9
1-5	168,496	267,328,200	609	1,185,500	80.4	78.6
6	28,207	43,838,200	161	269,700	81.0	76.3
7	27,119	41,929,600	183	231,100	66.2	64.6
8	26,238	40,422,200	137	230,000	66.6	62.5
9	25,380	38,739,100	124	235,100	58.9	62.8
10	24,609	37,473,200	139	219,400	64.2	56.7
6-10	131,553	202,402,300	604	1,185,300	67.2	64.4
11	19,701	29,225,500	116	233,700	63.5	72.7
12	16,282	23,882,100	104	226,900	64.6	85.9
13	13,185	19,009,300	117	172,900	83.7	73.4
14	10,465	15,969,000	97	168,900	82.7	79.3
15	8,089	11,587,300	77	123,700	78.6	73.8
11-15	67,712	98,766,200	511	926,100	73.1	77.1
16	5,639	7,918,800	42	59,900	57.6	49.2
17	3,906	5,506,900	23	24,000	42.0	26.6
18	2,407	3,230,800	25	49,300	66.1	80.3
19	1,061	1,489,200	7	9,500	39.1	32.1
16-19	13,012	18,204,700	97	142,700	52.9	47.1
11-19	80,724	116,970,900	608	1,068,800	68.9	71.1
6 & ov.	212,277	319,373,200	1,302	2,254,100	68.0	67.4
Total	380,773	686,701,400	1,993	3,439,600	71.9	70.9

Ages at Issue	TABLE 2(a)					
	Summary by Ages, Policy Year One					
15-19	2,577	3,472,400	4	5,000	59.7	55.4
20-29	16,368	21,406,200	54	89,000	113.6	122.6
30-39	11,996	19,355,600	14	15,500	37.3	25.5
40-49	6,477	13,360,700	24	47,500	80.0	76.4
50-59	1,799	4,482,800	5	6,500	31.5	14.1
60 & ov.	170	445,500	---	---	---	---
Total	30,387	62,522,200	101	153,500	71.7	63.5

Strong-Weisse—Women as Insurance Risks 323

TABLE 4—Continued—Issues of 1909 to 1918.

Policy Years One-Five.

Ages at Issue	Exposed to Risk		Died		Ratio of Actual to Expected	
	Number	Amount	Number	Amount	No. %	Amt. %
15-19	10,681	\$ 14,465,700	37	\$ 48,200	101.2	97.3
20-29	68,372	88,599,700	249	325,000	96.9	97.6
30-39	52,424	83,878,600	200	300,500	88.5	82.6
40-49	28,358	58,649,000	132	368,000	65.3	87.7
50-59	7,807	19,775,200	67	180,500	59.0	46.4
60 & ov.	764	1,960,000	6	13,300	25.0	21.7
Total	168,496	267,328,200	691	1,185,500	80.4	78.6

Policy Years Six-Ten.

15-19	8,071	10,706,200	21	20,500	60.3	44.0
20-29	52,555	65,866,100	206	240,700	85.7	79.9
30-39	41,902	64,071,700	167	235,800	62.4	58.1
40-49	22,362	44,253,200	187	447,700	65.2	78.6
50-59	6,071	14,920,000	91	188,700	54.4	45.2
60 & ov.	592	1,594,500	22	56,900	62.4	59.4
Total	131,553	202,402,300	694	1,185,300	67.2	64.4

Policy Years Eleven and Over.

15-19	4,665	5,927,900	12	13,500	57.4	50.8
20-29	31,923	38,379,700	115	130,500	68.1	64.2
30-39	27,104	39,017,200	204	293,000	81.8	80.8
40-49	13,235	24,759,900	163	324,700	61.8	65.1
50-59	3,485	8,129,100	91	227,300	60.3	66.1
60 & ov.	307	757,100	23	79,800	80.5	115.7
Total	80,724	116,970,900	608	1,068,800	68.9	71.1

Policy Years Six and Over.

15-19	12,736	16,724,100	33	34,000	59.2	46.5
20-29	84,483	104,245,800	321	371,200	78.4	73.5
30-39	69,006	103,985,900	371	525,800	71.7	67.5
40-49	35,597	69,018,100	350	772,400	63.6	72.3
50-59	9,556	23,049,700	182	411,000	57.2	54.8
60 & ov.	809	2,351,600	45	136,700	70.5	83.0
Total	212,277	319,373,200	1,302	2,254,100	68.0	67.4

All Policy Years Combined.

15-19	23,417	31,189,800	70	82,200	75.8	67.0
20-29	152,855	192,845,800	570	606,200	85.5	83.1
30-39	121,439	187,867,800	571	829,300	76.9	72.3
40-49	63,665	127,662,100	482	1,140,400	64.1	76.7
50-59	17,453	42,324,900	249	541,500	57.7	52.5
60 & ov.	1,663	4,311,600	51	150,000	58.0	66.4
Total	380,773	586,701,400	1,993	3,439,600	71.9	70.9

TABLE 4 (Continued)

Issues of 1919 to 1927 Inclusive.

Exposed from 1919 to 1928.

Women Only.

Summary by Policy Years, All Ages Combined.

Ages at Years	Exposed to Risk		Died		Ratio of Actual to Expected	
	Number	Amount	Number	Amount	No. %	Amt. %
1	143,934	\$ 287,090,400	261	\$ 642,800	51.6	58.4
2	108,062	215,594,400	308	564,800	58.8	48.8
3	85,714	169,956,000	244	613,100	53.7	60.2
4	63,825	124,918,000	228	455,000	62.7	55.4
5	46,790	91,257,800	156	371,900	53.8	55.6
1-5	448,325	888,815,600	1,197	2,647,600	56.0	55.5
6	32,851	61,872,800	107	265,200	47.6	53.3
7	21,733	39,067,600	73	117,500	47.3	36.7
8	13,094	24,262,500	37	64,500	38.4	31.2
9	5,812	10,420,000	27	49,500	50.1	51.2
6-9	73,550	136,522,900	244	497,000	46.8	44.3
Total	521,875	1,025,338,500	1,441	3,144,600	54.2	53.4

Ages at Issue	TABLE 4(a)					
	Summary by Ages, Policy Year One					
10-14	3,206	5,315,000	2	11,000	27.1	90.0
15-19	13,604	19,704,400	14	19,000	39.6	37.1
20-29	59,964	98,171,100	93	137,600	53.7	48.4
30-39	38,961	86,990,000	84	175,000	68.8	64.0
40-49	20,840	53,961,300	42	227,200	43.6	90.6
50-59	6,705	20,574,900	24	71,000	40.5	33.7
60 & ov.	654	2,373,700	2	2,000	16.2	4.5
Total	143,934	287,090,400	261	642,800	51.6	58.4

Policy Years One-Five.

10-14	6,631	10,103,000	6	17,000	36.9	62.3
15-19	30,959	58,454,700	51	68,000	39.0	35.5
20-29	186,421	296,290,600	379	591,500	56.0	54.6
30-39	125,070	274,925,000	388	636,700	65.4	55.9
40-49	67,253	170,353,800	208	790,400	59.5	69.3
50-59	21,501	68,499,500	131	453,000	45.3	48.0
60 & ov.	2,000	8,189,000	24	91,000	41.9	37.7
Total	448,325	888,815,600	1,197	2,647,600	56.0	55.5

Strong-Weisse—Women as Insurance Risks 325

TABLE 4—Continued—Issues of 1919 to 1927.

Policy Years Six-Nine.

Ages at Issue	Exposed to Risk		Died		Ratio of Actual to Expected	
	Number	Amount	Number	Amount	No. %	Amt. %
10-14	---	---	---	---	---	---
15-19	5,700	\$ 7,819,200	13	\$ 17,500	53.5	62.5
20-29	31,912	48,269,100	83	134,000	57.8	61.6
30-39	21,473	43,776,300	61	106,700	47.5	40.5
40-49	11,062	26,788,900	53	84,800	40.7	27.1
50-59	3,155	8,461,200	29	136,000	36.2	62.7
60 & ov.	258	1,408,200	5	18,000	34.0	23.2
Total	73,550	136,522,900	244	497,000	46.8	44.3

All Policy Years Combined.

10-14	6,031	10,103,000	6	17,000	36.9	62.3
15-19	45,650	66,273,900	64	85,500	41.2	38.0
20-29	218,333	346,569,700	462	725,500	56.3	55.8
30-39	146,543	318,701,300	399	743,400	61.9	53.0
40-49	78,305	197,142,700	321	875,200	55.3	60.2
50-59	24,746	76,960,700	160	580,000	43.3	50.7
60 & ov.	2,258	9,597,200	29	109,000	40.3	34.2
Total	521,875	1,025,338,500	1,441	3,144,600	54.2	53.4

TABLE 5
Effects of Occupation on Mortality—United States and Canada—Issues of 1907 to 1924, Exposed to 1925—Women Only

Class	Occupations	Entrants		Terminated otherwise than by Death		Percent. of total Entrants		Exposures		Actual		Ratios	
		No.	Amt.	No.	Amt.	No.	Amt.	No.	Amt.	No.	Amt.	No.	Amt.
1	General Exper. Male & Female					33.3	31.5					80.9	83.7
	15-29					33.1	37.4					87.5	90.6
	30-44					31.8	31.5					80.7	85.7
	45 & Over					24.5	24.2					77.0	80.3
Actresses	All Ages	186	\$1,340,500	56	\$346,500	30.1	25.8	838	\$5,309,000	5	\$6,500	130.5	28.5
	15-29	121	1,193,500	31	240,500	25.6	23.7	238	3,992,500	1	1,000	40.5	6.6
	30-44	124	1,147,000	25	106,000	29.1	32.7	10	1,411,000	3	4,500	209.8	59.3
	45 & Over	64	339,500	—	—	—	—	16	1,411,000	1	1,000	40.6	6.6
1	All Ages	181	418,600	58	153,000	32.0	36.6	765	1,629,100	6	7,500	93.0	52.6
	15-29	26	59,500	10	30,500	38.5	61.3	76	129,500	—	—	—	—
	30-44	99	237,500	31	84,500	31.3	35.6	446	960,500	2	2,000	79.1	39.2
	45 & Over	56	121,600	17	38,000	30.4	31.3	244	530,100	4	5,500	108.3	63.3
3	Chiropractors & Osteopaths					32.9	34.2	112,955	163,642,900	360	550,000	65.6	60.4
	All Ages	25,990	39,870,500	6,300	9,629,000	23.9	24.2	70,344	60,535,500	191	253,500	70.3	77.6
	15-29	16,488	21,153,300	3,004	3,674,000	29.1	20.3	38,546	27,000,000	122	217,000	67.6	66.3
	30-44	8,042	14,403,700	1,568	3,074,700	19.8	22.9	5,978	17,491,100	37	56,100	43.2	31.4
	45 & Over	1,460	4,393,500	306	955,900	21.2	22.9	4,288	10,270,600	20	34,400	53.0	36.6
4	Dressmakers-Proprietresses					31.3	41.9	737	1,181,000	—	—	—	—
	All Ages	804	1,677,300	231	550,800	28.7	32.8	4,288	10,270,600	20	34,400	53.0	36.6
	15-29	150	259,000	47	108,500	31.3	41.9	737	1,181,000	—	—	—	—
	30-44	446	888,700	133	330,500	29.8	36.1	2,485	5,566,000	8	17,000	52.5	47.6
	45 & Over	208	529,600	51	121,800	24.5	23.0	1,066	3,525,000	12	17,400	73.8	32.5
5	Factory Workers					27.2	25.6	5,968	6,138,400	27	24,500	39.8	80.0
	All Ages	1,468	1,583,100	400	408,700	27.2	25.6	5,968	6,138,400	27	24,500	39.8	80.0
	15-29	981	1,026,000	304	307,200	31.0	22.9	3,438	3,154,300	18	17,000	122.0	121.3
	30-44	408	474,100	81	93,500	20.0	19.7	1,430	1,971,100	6	9,000	56.8	46.0
	45 & Over	84	83,000	15	16,000	17.9	18.2	468	429,500	3	2,500	55.2	42.7
6	Farming					36.0	39.2	5,806	13,962,100	45	90,000	79.6	64.3
	All Ages	1,130	2,843,400	407	1,113,600	36.0	39.2	5,806	13,962,100	45	90,000	79.6	64.3
	15-29	165	298,000	55	111,000	33.3	37.2	854	1,441,000	2	2,500	57.3	43.0
	30-44	580	1,560,700	231	626,800	39.8	40.2	2,936	7,545,400	15	39,000	80.4	83.4
	45 & Over	385	984,700	121	375,900	31.4	38.2	2,016	4,971,700	28	46,500	81.5	55.4

TABLE 5 (Continued).
Effects of Occupation on Mortality—Women Only (Continued).

Class	Occupations	Entrants		Terminated otherwise than by Death		Percent. Terminated of total Entrants		Exposures		Actual		Ratios	
		No.	Am't.	No.	Am't.	No.	Am't.	No.	Am't.	No.	Am't.	A. M.	M. A.
7	Hotels-Boarding Houses Restaurants and Clubs Proprietresses	1,490	3,264,900	484	1,138,200	32.5	33.8	6,915	13,614,800	58	116,300	88.9	91.5
		220	392,000	82	104,500	36.3	28.9	809	1,199,500	4	4,000	120.6	87.3
		762	1,771,400	258	643,100	33.9	36.3	3,654	7,084,100	25	52,500	109.1	121.3
8	Housewives	602	1,231,500	144	390,700	23.7	31.7	2,452	5,331,200	29	59,800	74.0	75.5
		40,750	86,146,600	11,429	21,409,500	26.0	26.0	201,172	332,075,000	1,110	2,503,500	76.6	86.5
		14,355	23,243,100	4,546	7,635,800	33.7	32.9	66,901	103,891,500	239	436,300	111.4	106.5
9	Merchants	19,785	43,046,600	5,236	10,632,600	26.5	24.8	100,558	200,924,000	470	834,500	73.6	71.3
		6,610	19,899,900	1,344	3,151,400	20.3	16.0	33,768	87,560,100	380	1,292,700	64.9	94.5
		2,888	5,941,100	700	1,676,100	29.4	28.2	12,089	27,090,200	61	184,500	62.1	80.4
10	Nurses-Trained	3,371	804,600	123	302,500	33.2	35.5	1,713	3,202,500	6	15,000	87.8	119.4
		1,379	3,167,000	404	898,100	29.3	29.4	7,259	14,854,500	31	52,000	67.3	57.9
		632	1,970,100	173	470,500	27.3	23.9	3,117	9,003,200	24	137,500	62.9	92.4
11	Physicians	6,127	10,270,000	1,883	3,025,900	30.4	29.5	27,883	41,347,000	96	126,800	66.3	60.4
		3,410	5,476,100	1,179	1,880,700	34.6	34.3	13,940	20,165,900	63	63,600	96.6	81.2
		2,331	4,132,500	599	998,700	25.7	24.2	12,231	18,765,600	37	54,700	83.6	53.7
12	Printers	386	661,400	85	146,500	22.0	22.1	1,642	2,456,700	6	7,500	25.7	26.8
		103	607,900	38	106,000	19.7	17.4	1,972	3,299,900	7	18,700	55.7	60.3
		114	33,000	5	15,000	35.7	45.5	96	184,000	7	7,000	58.7	60.4
13	Saleswomen	107	350,300	19	56,000	17.8	15.6	818	1,944,500	3	11,700	56.9	79.9
		72	215,600	14	35,000	19.4	16.2	458	1,141,100	4	11,700	56.9	79.9
		111	132,600	27	25,000	24.3	21.1	568	611,100	8	8,600	219.8	237.5
14	Saleswomen	71	85,500	25	25,000	35.2	30.4	303	824,000	3	3,000	250.0	240.8
		32	40,600	2	2,000	6.3	4.9	188	229,100	3	3,600	236.2	296.3
		8	6,900	---	---	---	---	67	67,600	2	2,000	170.9	166.5
15	Saleswomen	3,553	4,538,400	1,071	1,390,900	30.1	29.1	15,558	18,675,000	54	59,100	65.5	59.4
		1,940	2,308,300	70	826,500	35.9	35.8	8,683	8,982,700	22	22,700	70.2	65.7
		1,343	1,880,100	313	421,000	23.3	22.4	6,296	8,117,700	28	31,900	80.1	72.6
16	Saleswomen	255	350,000	57	73,400	22.4	21.0	1,179	1,675,200	4	4,500	24.8	21.5
		255	350,000	57	73,400	22.4	21.0	1,179	1,675,200	4	4,500	24.8	21.5
		255	350,000	57	73,400	22.4	21.0	1,179	1,675,200	4	4,500	24.8	21.5

TABLE 5 (Continued).
Effects of Occupation on Mortality—Women Only (Continued).

Class	Occupations	Entrants		Terminated otherwise than by Death		Percent Terminated of total Entrants		Exposures		Actual		Ratios	
		No.	Amt.	No.	Amt.	No.	Amt.	No.	Amt.	No.	Amt.	A. M.	M. A.
14	Servants—Domestic and Private Families	938	1,149,200	295	392,700	29.8	26.3	3,992	4,493,600	26	32,500	127.9	144.7
		577	648,700	196	215,700	34.0	33.3	2,300	2,445,600	10	15,000	134.4	140.9
		133	153,000	43	50,000	32.3	33.3	1,383	1,445,000	7	11,000	132.4	140.9
		98	121,000	13	18,500	13.1	15.3	383	463,000	7	5,900	146.4	153.5
15	Students	5,294	8,974,300	1,068	2,073,200	22.1	23.1	24,086	37,402,100	58	78,200	64.9	55.9
		5,211	8,898,300	1,148	2,029,200	22.0	23.0	23,776	36,929,600	57	75,200	65.0	56.0
		67	115,000	19	43,000	28.4	37.4	258	453,500	1	3,000	73.0	141.7
		6	21,000	1	1,000	16.7	4.8	22	88,000	---	---	---	---
16	Teachers	20,548	30,465,000	4,294	6,007,500	20.8	19.7	98,731	132,107,500	310	412,500	60.0	53.9
		12,789	17,151,000	2,572	3,493,000	20.3	23.0	58,381	82,358,000	187	241,500	61.9	69.9
		14,259	10,694,000	1,722	2,514,500	12.0	16.3	33,969	42,389,200	112	166,000	53.9	69.9
		1,508	2,712,100	241	457,600	16.0	18.9	6,768	11,394,000	55	74,200	57.8	48.0
17	Telephone and Telegraph Operators	1,156	1,384,800	382	441,300	33.0	31.9	4,068	5,034,700	19	21,000	100.3	96.5
		873	981,800	321	362,300	36.8	36.9	3,298	3,546,700	14	13,500	113.7	101.5
		253	368,000	52	63,500	20.2	17.3	1,035	1,353,500	4	6,500	76.3	96.4
		25	35,000	9	15,500	36.0	44.3	106	134,500	1	1,000	71.4	56.1
Total	17 Occupation Classes	112,847	200,707,300	29,069	46,821,900	25.9	24.8	527,393	876,613,500	2,279	4,394,200	72.6	75.5
		57,618	88,743,100	16,599	26,939,400	28.8	28.3	267,156	419,575,500	1,022	1,927,000	82.2	80.5
		45,829	111,964,200	12,470	19,882,500	27.4	27.0	241,622	386,707,500	889	1,560,000	72.1	73.9
		12,298	38,217,500	2,994	6,327,600	21.1	19.0	59,949	145,921,900	567	1,794,400	62.1	75.5

Strong-Weisse—Women as Insurance Risks 329

TABLE 6.

Issues of 1907 to 1922, Exposed to 1924.

Policies terminated by death from principal causes among men and women respectively, with the death rate per 100,000 exposed to risk.

Policy Years One-Seventeen.

Cause of Death	Sex	Policies terminated by death				Death rate per 100,000 exposed to risk			
		All Ages	15 to 29	30 to 44	45 & Ov.	All Ages	15 to 29	30 to 44	45 & Ov.
Typhoid Fever	Men	604	301	220	83	11	14	9	10
	Women	41	30	9	2	9	15	5	4
Influenza-Epidemic	Men	2,937	1,581	1,159	197	55	74	50	23
	Women	191	125	59	7	41	57	31	13
Influenza-Endemic	Men	395	73	176	146	7	3	8	17
	Women	28	13	6	9	6	6	3	17
Tuberculosis	Men	2,301	1,073	891	337	43	50	38	40
	Women	199	113	69	17	43	52	36	31
Cancer	Men	2,171	157	743	1,271	41	7	32	151
	Women	319	16	163	140	69	7	86	253
Diabetes	Men	418	93	162	163	8	4	7	19
	Women	38	14	10	14	8	6	5	26
Apoplexy, Paralysis & Softening of Brain	Men	1,918	63	530	1,325	36	3	23	157
	Women	89	9	34	46	19	4	18	85
General Paresis of Insane & Insanity	Men	379	63	184	132	7	3	8	16
	Women	21	4	13	4	5	2	7	7
Heart Disease	Men	2,067	155	632	1,300	39	7	27	154
	Women	105	12	38	55	23	5	20	101
Angina Pectoris	Men	967	19	255	693	18	1	11	82
	Women	18	1	---	17	4	---	---	31
Arterio-Sclerosis	Men	317	5	78	234	6	---	3	28
	Women	7	---	1	6	2	---	1	11
Pneumonia	Men	2,577	757	951	869	49	35	41	103
	Women	153	44	67	42	33	20	35	77
Diseases of Liver and Gall Bladder	Men	509	55	207	247	10	3	9	29
	Women	55	10	30	15	12	5	16	28
Appendicitis and Peritonitis	Men	1,088	395	470	223	21	19	26	26
	Women	89	47	31	11	19	22	16	20
Bright's Disease	Men	1,766	163	627	976	33	8	27	116
	Women	91	11	44	36	20	5	23	66
Suicides	Men	1,148	224	567	357	22	10	24	42
	Women	24	15	6	3	5	7	3	6

TABLE 6 (Continued).
Policy Years One-Seventeen.

Cause of Death	Sex	Policies terminated by death				Death rate per 100,000 exposed to risk			
		All Ages	15 to 29	30 to 44	45 & Ov.	All Ages	15 to 29	30 to 44	45 & Ov.
Homicides	Men	272	86	141	45	5	4	6	5
	Women	6	4	2	---	1	2	1	---
Automobile Accidents	Men	600	236	323	127	13	11	14	15
	Women	29	11	11	7	6	5	6	13
Other Casualties	Men	2,465	1,009	909	487	47	47	42	53
	Women	57	24	23	10	12	11	12	13
The Puerperal State	Women	168	115	53	---	41	53	28	---
Total from All Causes	Men	30,613	7,939	11,415	11,269	678	372	491	1,337
	Women	2,106	742	828	530	466	340	437	999

DR. PATTON—The New York Life has enough information in its files to aid in the solution of any of our selection problems. Dr. A. B. Hobbs of that office will open the discussion of this paper.

DR. HOBBS—Since the Medico-Actuarial report was published we have not had any extensive investigation into the mortality among women. We are therefore greatly indebted to Dr. Weisse and Mr. Strong for their contribution to the literature on the subject. Few companies have apparently made recent investigations into the mortality among women, as the general impression seems to be that it is better than among men. Early in this paper there is a reference to pregnant women. The authors state that in 1908 their Company began to take such risks up to the seventh month—provided they had had a previous normal labor. Our Company postpones all these cases until the applicant has fully recovered from the incident. It would have been of interest to have included the different methods of treating these cases by the various Companies.

When we study the tables presented in this paper we note that the results obtained by the collaborators are on the same lines as in the M. A. report. There is one rather striking difference, namely that the mortality in class 4—widows and divorcees—was not only distinctly better than among married women as in the

Discussion—Women as Insurance Risks 331

M. A. but even better than among self-supporting spinsters. The amount of the material indicates that this very favorable mortality is not due to accidental fluctuation but the authors do not comment on this result.

It seems to me that this is a case where the age distribution is responsible for misleading results. In the M. A. the ratio of actual to expected deaths by age group at entry was not materially different in the classes of married women and widows and divorcees. About 60% of the deaths, however, were concentrated at entry ages under 40 in the case of married women, where the ratio of actual to expected runs from 120% to 150%, whereas in the case of widows and divorcees about 2/3 of the deaths occurred among entrants at ages 40 to 60 where the ratio of actual to expected runs about 105%.

A similar situation exists in the case of the paper under discussion. Compare Class 1(a), self-supporting spinsters with Class 4, Widows and Divorcees, in Table 3, issues of 1907 to 1922 exposed to 1924, all policy years combined. Omitting groups with less than 25 deaths—for the purpose only of illustrating and calling attention to the results of age distribution, we have the following:

Age Group at Entry	Class 1(a)			Class 4		
	No. of Deaths	Ratio	Act. Exp. by No.	No. of Deaths	Ratio	Act. Exp. by No.
15-19	33		76
20-29	352		76
30-39	186		65	87		80
40-49	105		61	126		68
50-59	34		47	108		58
60 & over				43		76
All ages			68		67

Thus it will be seen that for all ages combined Class 4 shows a slightly lower mortality ratio than Class 1(a) though in each age group the ratio is higher.

With regard to the mortality by occupation, no satisfactory conclusion can be drawn in a number of the classes on account of the limited amount of data. For example, among a class of actresses there were only five deaths, among chiropractors and

osteopaths six deaths, among physicians seven deaths and among printers eight deaths. On the other hand there was substantial material in such groups as clerical workers, housewives and teachers, all of whom showed a very satisfactory mortality on the whole. I assume that where the mortality was high at the younger ages the authors of the paper would not expect the companies to discriminate against them, realizing that the higher mortality at the younger ages would be more than offset by the better mortality at the older ages.

It is interesting to note that although there is a general opinion that the wives of farmers may not be good risks, the mortality was satisfactory. It is also gratifying to note that the mortality among trained nurses was low, although the experience of more than one company has shown that the rate of disability is very high among them. It would add to our knowledge if the authors could make an investigation separately of the trained nurses who are engaged in sanatoria for the tuberculous. The mortality among teachers is favorable and fairly even in all age groups. When, however, we study our claims for disability benefits we are inclined to believe that the morbidity is much above the general average. When our experience justifies it, we will be obliged to study risks not only from the standpoint of mortality but also from that of morbidity—two entirely different phases of our life insurance work.

DR. PATTON—The growth or development of any organization depends upon its absorption of new ideas. New members are essential to our continued prosperity and we are pleased to have a discussion of this paper by Dr. George Cullen of the Illinois Life.

DR. CULLEN—Mr. President and Gentlemen: The valuable statistics given us by Dr. Weisse were read by me and the first thing that occurred to me was to attempt to interpret the findings of those statistics. Was there any explanation of the mortality that was shown? In general we know that the mortality of females, except for one age group, that is between 15 and 19 years of age, is more favorable than the male group.

Discussion—Women as Insurance Risks 333

While the insurance experience has been satisfactory, there are certain points that should be taken into consideration in our selection of female risks. Of first importance is a consideration of the reasonableness of a woman applying for insurance. The wife who applies for insurance and makes her husband the beneficiary—and especially is this true if the husband does not carry an equal or greater amount in her favor—should give us pause in the approval of such an application. Where the husband does not carry insurance, I think that should be reason for declination of the application.

Mortality in women is affected as much by the social condition of the woman as any other element in her life. There are more ways of killing a woman than by ordinary disease. For instance, the wage earner is also the home maker, which puts an added burden on her life and makes her a less acceptable risk for the very reason of her feeling of responsibility to her family. So I say that occupation as occupation alone is quite as hard to determine and hard to evaluate as anything I have found in the medico-actuarial report on occupational mortality just published.

The cancer incidence in female risks is with us and will always account for a certain number of deaths. The deaths due to parturition in the United States is something that the medical profession has reason to be ashamed of, in that we are almost at the top of the list of mortality suffered at time of confinement. Dublin, gathering together as many statistics as he was able to obtain, states that on the basis of live births for 1920 our mortality death rate was about 8 per thousand while that of the Netherlands was only 2.4, that of Sweden 2.6, etc. Scotland, France and Belgium all show results which are more favorable. That is something we can look forward to with the hope that this mortality at least can be improved.

DR. PATTON—The next speaker had agreed to tell us about his company's experience with women as life insurance risks, but informed me some time ago that their material would not be reviewed in time for our meeting. He then agreed to give us his personal impressions, but was unfortunately called away and

what Dr. Scholz had for us will be given by Dr. Snow of the Massachusetts Mutual.

DR. SNOW (*reading paper of Dr. Scholz*)—The experience of our Company in the selection of female risks is comparable with the Mutual Life in that we also offered insurance to women from the organization of our Company in 1851, and apparently with little restrictions in the earlier years.

We expected that mortality studies the Massachusetts Mutual was making would be completed by this time, and had hoped to be able to present some figures of the Company's female experience. The data are in compilation but not yet in condition to present. The only definite figures we have are those presented to this Association about ten years ago, in which a gross mortality for all ages was 207% of the Company's normal experience, 166% of the M. A. table. The figures are also comparable to those shown by Dr. Weisse, in that the mortality experience of wage earning spinsters was approximately that of or slightly better than the Company's average, the great excess being on lives of married women. After this and other startling demonstrations, our rules on the acceptance of women were materially changed. Slight modification from time to time had been made. Our present consideration of female lives is as follows:

Applications from single women will be considered on any plan.

Applications from business or professional women, single or married, will be considered on any plan.

If a married woman has a life interest in an estate; that is to say an interest which produces a regular income during her lifetime, but which terminates upon her death, and which she cannot dispose of by will; or the insurance is desired to cover inheritance taxes; or her husband is uninsurable, she may be considered for an individual policy on any plan.

Applications for joint policies from husband and wife when there are living children will be considered on any plan except Term for an amount not in excess of \$2,500.

Discussion—Women as Insurance Risks 335

Any disability benefits granted in connection with policies issued to women since January 1, 1923, or under any case where the current disability features are attached to policies that were originally issued prior to January 1, 1923, will cease upon marriage.

If, however, the insured is already in receipt of disability benefits at the time of marriage, the payments will be continued until her death or recovery.

We all recognize that conditions have materially changed. Prior to 1910 insurance on wage earning women was inconsiderable as a group. Since this time there has been a progressively larger number of women wage earners. More women have engaged in the professions, and there has been an increasing number of women agents, from whom we feel we receive the best class of business on female lives.

Speculation probably still obtains, but in a much less degree than twenty or thirty years ago. Our examiners are of better type and in my judgment, better history takers. Obstetrical and gynecological service has radically improved in recent years.

Inspection service has greatly improved. Women's home life is not so confining. They are more active, dress more sensibly and have better hygiene in every way than their mothers. Publicity regarding tuberculosis, cardio-renal disease, cancer, and health examination are all beneficial factors. All of these factors should assure lower morbidity and mortality.

We realize that this paper relates to insurance and not to disability, but the inter-relation is so strong we cannot overlook the fact that for some years our disability experience on lives of women risks was materially higher than that on men, $2\frac{1}{2}$ to 1 on exposures. Whether chance or better selection, or both, is responsible, disability claims in the last two years show a very much better proportion. We are naturally hopeful that this will continue.

DR. PATTON—Through our years of experience in this body, we have met no more earnest worker nor one more willing to give of his time and talents for our benefit than Dr. Henry A.

Baker of the Kansas City Life, who will now address us. Dr. Baker.

DR. BAKER—Dr. Weisse and Mr. Strong in their paper on "Women as Life Insurance Risks" have given us one of the most interesting and valuable contributions to the literature of life insurance that has ever been written. Dr. Weisse is an authority on the subject of women as life insurance risks. I well remember that just twenty-one years ago, in 1908, he gave us a table of standard weights for women, which has been accepted ever since as the best table we have. I had the honor of discussing his paper at that time. He now gives us proof of what we have long surmised, namely, that women are undoubtedly better life insurance risks than men. The gentlemen who have preceded me have discussed this matter so thoroughly that little remains to be said. However, I wish to express to Dr. Weisse our great appreciation of his paper and, so far as our own office is concerned, we will immediately put into effect the conclusions arrived at and will liberalize our selection of female risks.

It is undoubtedly a fact that, in the old days when an extra premium was charged and female risks were accepted by the companies only with reservation, the mortality was higher. Any class so selected against will show a higher mortality. The very fact of imposing an extra premium increases the selection against the company by both applicants and agents. Under a free and unrestricted solicitation in this class a larger proportion of the best risks will be secured and the mortality will correspondingly decrease.

Dr. Weisse's rules with regard to the acceptance of pregnant women are liberal and we would be greatly interested to know the experience of his company with risks in this class. I believe statistics will show that the mortality from childbirth in our best lying-in hospitals is less than one-half of one per cent, and probably the mortality among women who are confined at their homes is even less, as the hospitals, of course, get the more serious cases. When we consider that this slight extra mortality is more than balanced by better habits, less hazardous occupa-

tion, and better modes of living among the women than among the men, we are inclined to believe that pregnant women can be accepted, if multiparae at standard rates and without any extra premium. However, we should like to have the doctor's opinion on this subject.

There is one class of women that showed a higher mortality in the Medico-Actuarial Investigation, namely, the married women under age thirty who had had no children. The experience in this class was 147%. It is interesting to note that this same class in the experience of the Mutual Life gave a mortality 23% in excess of the men. This shows that the same adverse factors in this class are still working, chief among which is the moral hazard or selection against the company. A certain number will always be insured in this class where the applicant or her husband, or both, are aware of unfavorable factors which are not given to the company, and which make insurance on her life very desirable to the husband.

In our own company, we freely accept unmarried women and widows up to age forty-five for \$5,000 or less without a medical examination on all forms and without an extra premium. I might say that while we have not reduced our experience to figures, we are firmly convinced that it is favorable on all classes of women risks.

In closing, I wish again to thank Dr. Weisse and Mr. Strong and to compliment them very highly on the thorough manner in which this subject has been handled.

DR. PATTON—Dr. Weisse, have you some closing remarks?

DR. WEISSE—Yes, doctor, I have. First I want to thank the four gentlemen who were kind enough to read my paper over and express their ideas about it.

In the first place, Dr. Hobbs brought up the question of having a lower mortality among older married women than among single women and widows. The only thing I can ascribe that to off hand is the probability of more careful inspection and consideration of the question of speculation and insurance hazard in that group. The point is made about our getting a good mortality

on farmers' wives. We did, but I did not say anything about the disability. I don't have to; you all know.

Dr. Cullen brought up the question of the insurance hazard. We follow that very carefully and have, if anything, a more thorough inspection possibly in the case of a woman applying for insurance. We have a special blank that they have to fill out as to what the husband's income is and what insurance he has.

Then there is also a point, I think, about women that does help the mortality, and that is the fact that the woman in the family gets earlier medical attention in case of illness than the man of the family. He has got to work as a rule, and if he feels under the weather, he goes out and works anyway. Unless the woman is a wage earner, she is more apt to let her husband know that she is sick and she wants to see the doctor.

There is one point that Dr. Baker made that I want to ask about. Did I understand you correctly, that you issued \$5,000 on women without medical examination, Dr. Baker? I didn't understand whether it was without medical examination or without special examination.

DR. BROWN (Dr. Baker being out of the room)—Without medical examination.

DR. WEISSE—I thank you gentlemen very much.

DR. PATTON—Gentlemen, I want to take this occasion to thank you all for the extreme courtesy with which you treated me as President of your association this year. I have enjoyed this meeting immensely because you have so answered the calls I made upon you in regard to this program to make me feel that I have good reasons to have enjoyed it.

In order that our coming President may have some experience before next year's meeting, I am going to ask Dr. Muhlberg to take the chair for the last paper.

DR. MUHLBERG—Before assuming my duties, I would like to express my thanks to you gentlemen on behalf of my company and myself for the honor which you conferred upon me. I of course appreciate the burdens and the difficulties of this office,

but with the assistance of our most efficient Secretary and my predecessors and fellow officers, and also the various committees, I hope that I will be able to carry on on the same high plane that we have had heretofore. At any rate, I promise to do my utmost with your very kind cooperation and I am hoping for the best.

I am afraid though that I am going to start out my service in a rather inauspicious way in that I am going to be immediately guilty of plagiarism. I have been particularly impressed with the way Dr. Patton has introduced the various speakers. I think it is a mighty fine idea, in introducing the speaker, to write down the remarks and then to read those remarks. In the first place, you say just exactly what you want to say and you can say it concisely. Secondly, you are not tempted to anticipate and steal all the glory of the speaker that is about to be introduced. I think the most pestiferous person in the world is the one who introduces you and tells the audience everything that you intended to tell.

I think before proceeding with the next paper, Dr. Piper has something to say.

DR. PIPER—There is a matter which, I am sure, will give us all unusual pleasure. We have enjoyed the hospitality of The Prudential Insurance Company and they have accorded us every facility to make possible a very helpful meeting. I know that we have enjoyed their auditorium, and that we have appreciated the bountiful and tasteful luncheons which they have provided and every attention which they have given. It therefore gives me pleasure to move that we express to them our profound appreciation of their hospitality in entertaining this convention.

Dr. Weisse seconded the motion.

DR. MUHLBERG—May I suggest that we vote in favor of that motion by a rising vote.

The motion was carried unanimously.

DR. MUHLBERG—Abnormal weights are with us each day of our office work. Delays in action are dangerous and any method

by which we can shorten the time without decreasing the accuracy of our information is valuable. We have had presented to us several ways of estimating the weight from other data in the reports. I was glad to welcome for this meeting the results of observations and calculations made at the Home Office of the Mutual Benefit by Dr. Charles P. Clark and Mr. John S. Thompson, who will submit the next paper.

DR. CLARK—Mr. President, I am going to invite these gentlemen to be my guests in the front seats, because I have some charts here. My discussion is going to be informal and largely based on these charts, and back there you can't see what I am talking about. I am going to explain first that this study was not the result of some marvelous idea which I had and which I thought might work out. Like most inventions and discoveries, so to speak, it resulted purely by accident.

(Dr. Clark proceeded to explain the charts he had prepared.)

CHEST AND ABDOMINAL MEASUREMENTS AS
RELATED TO HEIGHT AND WEIGHT, WITH
PRESENTATION OF TABLES OF AVERAGES.

CHARLES P. CLARK, *Medical Director, Mutual Benefit
Life Ins. Co.*

The successful underwriting of life insurance risks is dependent upon our ability to separate risks into various groups, the mortalities of which differ materially, making it possible for us to reject the less favorable classes. Such selection is more easily accomplished if we have before us tables of averages with which we may compare the findings in the individual.

The study which I wish to present relates to the question of build, and is I believe worthy of consideration in connection with the important work that has been done in the past and which will be done in the future in the study of weight and its influence on mortality.

Early in the selection of risks by our own Company, which was founded in 1845, an effort was made to ascertain whether an applicant was active or sedentary and whether he had a well formed chest. In the year 1865 for the first time a specific request was made of the Examiner to note the measurements of chest at inspiration and expiration, and it was not until 1882 that he was required also to secure the girth of abdomen. I believe we are safe in stating that few, if any, life insurance companies today would care to omit such measurements from their examination blank. The question then naturally arises as to the value of these measurements in insurance selection.

Without doubt the request for measurements of chest at inspiration and expiration was prompted by the desire to eliminate the tubercular risk. Later the recognition of an increased hazard in granting insurance to an applicant with

a protuberant abdomen was probably responsible for the added request for a measurement of abdominal girth.

The study which I here present is the result of an effort to develop a chart of averages in respect to circumferences of chest at inspiration and at expiration, also circumference of abdomen (or waist).

The development of a table of this kind was facilitated by the following observation:

I found that the abdominal or chest girth could not be estimated from height alone or weight alone. If, however, a group of risks are taken who average the same number of pounds per foot of height, it will be found that not only will they have approximately the same abdominal girths but it will also be found that their chest measurements very nearly correspond. For example, if three individuals have the following weights and heights, they will each average 29 pounds for each foot of height.

145 lbs.	159½ lbs.	174 lbs.
5' 0"	5' 6"	6' 0"

Notwithstanding the difference in heights and weights these men, if true to average, will have very similar chest and abdominal measurements. Let me illustrate with three such cases taken from the group of cards which I have at hand.

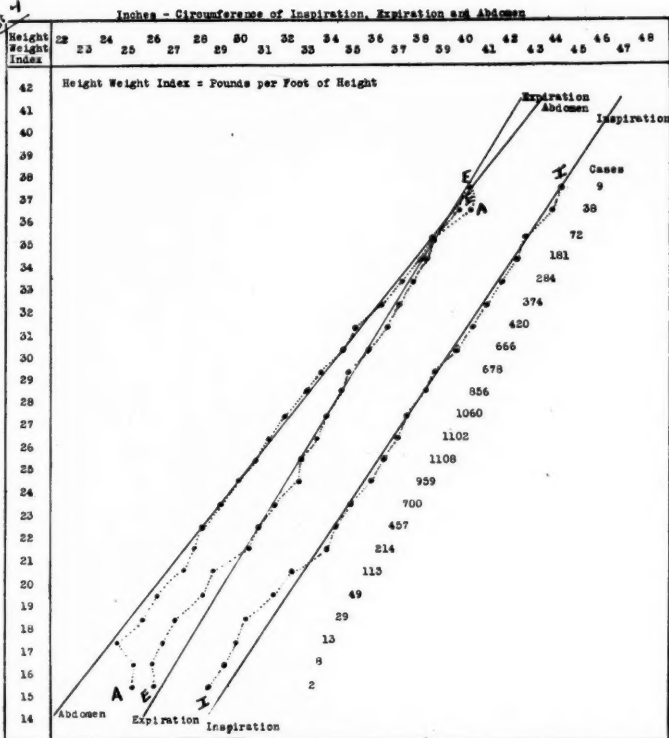
	Case I	Case II	Case III
Weight	145	159½	174
Height	5' 0"	5' 6"	6' 0"
Weight (in pounds) divided by Height (in feet.)	29 lbs.	29 lbs.	29 lbs.
Inspiration	37	37	37
Expiration	34	33	33
Abdomen	31	32½	31

Although individuals may differ considerably, yet an average can easily be obtained for any weight-height ratio.

A graph giving the results of a study of the measurements of 9,392 approved applicants is here given.

In the vertical column are given the number of pounds per foot of height (i.e.—weight divided by height).

6054



STUDY BASED ON 9,392 CASES

Above, in the horizontal line, are given circumferences in inches.

The 9,392 cases were sorted according to pounds per foot of height. Each group was studied separately, the average circumferences for inspiration, expiration and abdomen separately ascertained and the result indicated on the chart. For example, there were 1,108 cases which fell into the group averaging 25.4 pounds per foot of height. The average inspiration in this group was 36.4 inches, the average expiration 32.6 inches, and the average circumference of abdomen 30.6 inches. Having indicated on the graph the averages in the various groups, a line was so drawn that the minimum deviation was obtained. It was then noted that an individual averaging 41 pounds per foot of height should have an inspiration of 46.5 inches; and at the other end of the line drawn it was noted that an individual averaging 15 pounds per foot of height should have an inspiration of 29 inches. Therefore, for 26 pounds increase per foot of height there was an increase in circumference at inspiration of 17.5 inches, or for every pound the increase was 0.673 inches. This factor of increase was also obtained for expiration and abdomen, as noted in the following:

Factor of increase for each pound per foot of height:

Inspiration	0.673 inches
Expiration	0.614 inches
Abdomen	0.796 inches

This graph represents we believe the curve for ages 35 to 40. Men at older ages on the average have larger circumferences than those found in the table. It should be noted in the graph that the increase is quite uniform representing approximately a straight line after 22 pounds per foot of height is reached, but below that figure the actual is somewhat below the theoretical line for inspiration and expiration and above the line for abdomen. This is probably due to the fact that the individuals comprising groups 15 to 22 are for the most part boys and young men at or shortly

after the period of puberty. It has been recognized that it is during puberty that the chest development becomes most obvious.

Having thus obtained the factors for the increase of inspiration, expiration and abdomen, the theoretical measurements for pounds per foot of height were obtained as seen in the following table:

Pounds per Foot of Height	Inspiration	Expiration	Abdomen
	Inches	Inches	Inches
15	29.0	26.2	22.3
16	29.7	26.8	23.1
17	30.3	27.3	23.9
18	31.0	28.0	24.7
19	31.7	28.6	25.5
20	32.4	29.2	26.3
21	33.0	29.8	27.1
22	33.7	30.5	27.9
23	34.4	31.1	28.7
24	35.1	31.7	29.5
25	35.7	32.3	30.3
26	36.4	32.9	31.1
27	37.1	33.5	31.9
28	37.7	34.1	32.7
29	38.4	34.8	33.5
30	39.1	35.4	34.2
31	39.8	36.0	35.0
32	40.4	36.6	35.8
33	41.1	37.2	36.6
34	41.8	37.8	37.4
35	42.5	38.4	38.2
36	43.1	39.1	39.0
37	43.8	39.7	39.8
38	44.5	40.3	40.6
39	45.2	40.9	41.4
40	45.8	41.5	42.2
41	46.5	42.1	43.0
42	47.1	42.7	43.8

With these data we next proceeded to form the following scale of measurements for various heights and weights:

(See Photostat.)

In the first, second and third vertical columns are given the theoretical circumferences for inspiration, expiration and abdomen corresponding to individuals with any height and



INSP.	KLP.	ABDOMEN	TOTAL	HEIGHT 5'0"	1"	2"	3"	4"	5"	6"	7"
28.4	25.7	21.7	75.8	71							
28.7	25.9	22.0	76.6	73	75						
29.1	26.2	22.4	77.7	75	77	78					
29.4	26.5	22.5	78.7	78	79	81	82				
29.7	26.8	22.1	79.6	80	82	83	84	86			
30.1	27.1	23.3	80.7	83	84	86	87	88	90		
30.3	27.4	23.9	81.6	85	87	88	89	91	92	94	
30.6	27.7	24.3	82.6	87	89	90	92	93	95	96	98
31.0	28.0	24.7	83.7	90	91	93	94	96	97	99	100
31.3	28.3	25.1	84.7	92	94	95	97	98	100	101	103
31.7	28.6	25.5	85.8	95	96	98	99	101	102	104	106
31.9	28.8	25.8	86.5	97	99	100	102	103	105	107	108
32.2	29.1	26.1	87.4	99	101	103	104	106	108	109	111
32.6	29.4	26.6	88.6	102	103	105	107	108	110	112	113
32.9	29.7	26.9	89.5	104	106	107	109	111	113	114	116
33.2	29.9	27.2	90.3	106	108	110	112	113	115	117	119
33.4	30.2	27.6	91.2	109	110	112	114	116	118	120	121
33.8	30.6	28.0	92.4	111	113	115	117	119	120	122	124
34.1	30.8	28.4	93.3	113	115	117	119	121	123	125	127
34.5	31.2	28.8	94.5	116	118	120	121	124	125	127	129
34.7	31.5	29.2	95.4	118	120	122	124	126	128	130	132
35.0	31.7	29.5	96.2	120	122	125	126	129	131	132	134
35.4	32.0	29.9	97.3	123	125	127	129	131	133	135	137
35.7	32.3	30.3	98.3	125	127	129	131	134	136	138	140
36.1	32.6	30.7	99.4	128	130	132	134	136	138	140	142
36.4	32.9	31.0	100.3	130	132	134	136	139	141	143	145
36.7	33.2	31.4	101.3	132	134	137	139	141	143	145	148
37.0	33.5	31.9	102.4	135	137	139	141	144	146	148	150
37.3	33.8	32.2	103.3	137	139	142	144	146	148	151	153
37.6	34.0	32.5	104.1	139	142	144	146	149	151	153	156
38.0	34.4	33.0	105.4	142	144	146	149	151	153	156	158
38.3	34.6	33.3	106.2	144	146	149	151	154	156	158	161
38.6	34.9	33.6	107.1	146	149	151	154	156	159	161	163
39.0	35.2	34.0	108.2	149	151	154	156	159	161	164	166
39.2	35.5	34.4	109.1	151	154	156	159	161	164	166	169
39.5	35.7	34.7	109.9	153	156	159	161	164	166	169	171
39.9	36.1	35.2	111.2	156	158	161	164	166	169	171	174
40.2	36.3	35.5	112.0	158	161	163	166	169	171	174	177
40.4	36.6	35.8	112.8	160	163	166	168	172	174	177	179
40.8	36.9	36.2	113.3	163	166	168	171	174	176	179	182
41.1	37.2	36.6	114.9	165	168	171	173	177	179	182	184
41.5	37.5	37.0	116.0	168	170	173	176	179	182	184	187
41.6	37.8	37.4	117.0	170	173	176	178	182	184	187	190
42.1	38.1	37.8	118.0	172	175	178	181	184	187	189	192
42.5	38.4	38.2	119.1	175	177	181	183	187	189	192	195
42.7	38.7	38.5	119.8	177	180	183	186	189	192	195	198
43.0	38.9	38.9	120.8	179	182	185	188	192	194	197	200
43.4	39.3	39.3	122.0	182	185	188	191	194	197	200	203
43.7	39.5	39.6	122.8	184	187	190	193	197	199	202	205
43.9	39.8	40.0	123.7	186	189	193	196	199	202	205	208
44.3	40.2	40.5	125.0	189	192	195	198	202	205	208	211
44.6	40.4	40.8	125.8	191	194	198	201	204	207	210	213
44.9	40.7	41.2	126.8		197	200	203	207	210	213	216
45.3	41.0	41.6	127.9			202	206	209	212	215	219
45.6	41.3	42.0	128.9				209	212	215	218	221
46.0	41.6	42.4	130.6					214	217	221	224
46.2	41.8	42.6	130.6						220	223	226
46.5	42.1	43.0	131.6							226	229
46.8	42.4	43.4	132.6								232
47.2	42.7	43.8	133.7								
47.5	43.0	44.2	134.7								
47.8	43.3	44.6	135.7								
48.1	43.6	44.9	136.6								
48.4	43.8	45.2	137.4								
48.7	44.1	45.6	138.4								

6"	7"	8"	9"	10"	11"	6'0"	1"	2"	3"	4"	5"
94											
96	98										
99	100	102									
01	103	104	106								
04	106	107	109	110							
07	108	110	111	113	115						
09	111	112	114	116	117	119					
12	113	115	117	119	120	122	124				
14	116	118	120	121	123	125	126	128			
17	119	120	122	124	126	128	129	131	133		
20	121	123	125	127	129	130	132	134	136	138	
22	124	126	128	129	131	133	135	137	139	141	143
25	127	128	130	132	134	136	138	140	142	144	146
27	129	131	133	135	137	139	141	143	145	147	149
30	132	134	136	138	140	142	144	146	148	150	152
32	134	136	138	140	143	145	146	149	151	153	155
35	137	139	141	143	145	147	149	151	153	155	158
38	140	142	144	146	148	150	152	154	156	158	161
40	142	144	147	149	151	153	155	157	159	161	164
43	145	147	149	151	154	156	158	160	162	164	167
45	148	150	152	154	156	159	161	163	165	167	170
46	150	153	155	157	159	162	164	166	168	170	173
51	153	155	157	160	162	164	166	169	171	173	176
53	156	158	160	162	165	167	169	172	174	176	179
56	158	161	163	165	168	170	172	175	177	179	182
58	161	163	166	168	170	173	175	178	180	182	185
61	163	166	168	171	173	176	178	180	183	185	188
64	166	169	171	173	176	178	181	183	186	188	191
66	169	171	174	176	179	181	184	186	189	191	194
69	171	174	176	179	182	184	187	189	192	194	197
71	174	177	179	182	184	187	189	192	195	197	200
74	177	179	182	184	187	190	192	195	198	200	203
77	179	182	185	187	190	193	195	198	201	203	206
79	182	185	187	190	193	195	198	201	204	206	209
82	184	187	190	193	196	198	201	204	206	209	212
84	187	190	193	195	198	201	204	207	210	212	215
87	190	193	195	198	201	204	207	209	212	215	218
89	192	195	198	201	204	207	210	212	215	218	221
92	195	198	201	204	207	210	212	215	218	221	224
95	198	201	203	206	209	212	215	218	221	224	227
97	200	203	206	209	212	215	218	221	224	227	230
00	203	206	209	212	215	218	221	224	227	230	233
02	205	209	212	215	218	221	224	227	230	233	236
05	208	211	214	217	221	224	227	230	233	236	239
08	211	214	217	220	223	227	230	233	236	239	242
10	213	217	220	223	226	229	232	236	239	242	245
13	216	219	222	226	229	232	235	239	242	245	248
15	219	222	226	228	232	235	238	241	244	248	251
18	221	224	228	231	235	238	241	244	248	251	254
21	224	227	231	234	237	241	244	247	251	254	257
23	226	230	233	237	240	243	247	250	254	257	260
26	229	233	236	239	243	246	250	253	256	260	263
	232	235	239	242	246	249	252	256	259	263	266
	235	238	241	245	249	252	255	259	262	266	270
		244		248	251	255	258	262	265	269	273
				250	254	258	261	265	268	272	276
					257	260	264	268	271	275	279
						263	267	271	274	278	282
							270	273	277	281	285

any weight in the same line to the right. For example, an individual 5'0" in height who weighs 118 pounds should have theoretically an inspiration of 34.7 inches, expiration 31.5 inches, and abdomen 29.2 inches. The same measurements, theoretically, are found in individuals 5'1" in height weighing 120 pounds, 5'2" weighing 122 pounds, 5'3" weighing 124 pounds, etc. With these theoretical average measurements one can, as will be noted later, state how much the circumferences of any given individual vary from the theoretical average.

One value of such a table is that it affords a means of checking the stated weight in our overweight applicants. The method is as follows: Add the three measurements—chest at inspiration, chest at expiration, and abdomen. Find the corresponding figure in column four; proceed to the right to column corresponding to height; the figure at the intersection will be the estimated weight. This feature of the study appeared to be of so much importance that Mr. Thompson kindly consented to make a special study which he will present to the Association.

It might be well to explain, however, that if the estimated and stated weights do not agree one cannot be certain that the stated weight is incorrect and the estimated weight correct. The build of an individual may differ very materially from the average. We are all acquainted with the type that has enormous extremities and small torso. In such cases the weight would be underestimated. For the individual with a preponderance of fat accumulation over chest and abdomen the weight would be overestimated. In young individuals devoid of fat the weight may be underestimated and with advancing years and increasing adiposity the weight may be overestimated. However, from a practical standpoint, we have found that a marked discrepancy between the estimated and stated weights in an overweight is worthy of investigation. The majority of the estimated weights will be within ten pounds of the actual weight as Mr. Thompson will show later.

We have been using this chart for approximately a year and a half in checking cases and during that time we have uncovered some gross misstatements of weight. It has, therefore, been our custom to check gross discrepancies even in those cases where the Examiner has stated that he actually weighed the applicant. Of the large number of illustrative cases of which I have kept a record, I will cite only three.

J. C. C.—Age 42.

Height 6'0", Weight 200 lbs.—Stated to have been weighed.

Chest at Insp.	45"
Chest at Exp.	43"
Abdomen	43"

Estimated weight=243 pounds.

We wrote Examiner as follows:

"It is stated that applicant's height is 6 feet, weight 200 pounds. An estimate of applicant's weight based on the height and chest and abdominal measurements would indicate that applicant may be considerably in excess of this weight, and we are therefore writing to ascertain whether you, yourself, weighed the applicant and with ordinary clothing including coat and vest. If not, will you kindly favor us with applicant's exact weight while fully clothed. Does he appear to be of the obese type.

"We would much appreciate your filling out the enclosed overweight blank."

He replied that correct weight is 230 pounds and mistake was made in recording weight in examination blank. Mortality on stated weight is 115%; on correct weight 161%. We declined.

M.P. D.—Age 38.

Height 5'7", Weight 185 pounds. Stated to have been weighed.

Chest at Insp.	43"
Chest at Exp.	40"
Abdomen	39"

Estimated weight 204 pounds. The mortality on the original weight stated was 123%.

We wrote to our Examiner as follows:

"Applicant is described as stout and his height and weight are given as 5 feet 7 inches, 185 pounds.

"An estimate of his weight based on height, chest, and abdominal measurements would indicate that this party weighs 200 or more pounds. We therefore would appreciate a rechecking of applicant's weight with coat and vest."

The Examiner's reply was as follows:

"Your favor of the 30th ultimo received; sorry to have been so slow in answering but have had my office upset some in moving, hence the delay.

"This moving had caused my scales to get out of balance some four or five pounds, in fact they had got sprung where the weight was not accurate. However, I have had them re-checked and they are absolute now. Weighing Mr. Daley with his coat and vest on, the absolute weight is 203 pounds. I regret this error but do not think it will happen again.

"Thanking you for calling my attention to this and assuring you of my thorough cooperation at all times, I am."

The mortality based on the correct weight is 152%.

S. E. E.—Age 39.

Height and Weight 5' 10", 202 pounds. Stated to have been weighed.

Chest at Insp.	48"
Chest at Exp.	45"
Abdomen	43½"

Estimated weight—245 pounds plus.

We wrote the Examiner calling for a checking of applicant's weight on tested scales while fully clothed, including coat and vest.

The Examiner replied as follows:

"As you have requested I have checked applicant's weight and measurements. With coat and vest and clothing on he weighed 238 pounds. Stripped his weight is 232 pounds. That is exactly what his weight was at the time of the first exami-

nation. The record of 202 is an error on my part and should be 232. I regret very much that this mistake was made but I reckon we are all only human."

If applicant had weighed 202 pounds he would have been 35 pounds overweight with a mortality of 127%. Weighing 238 pounds, he is 71 pounds overweight with a mortality of 189%.

Explanations on the part of Examiners regarding the discrepancies have been varied, but many have admitted that they accepted the applicant's statement believing that they were entirely honest and trustworthy. In some cases the fault has been apparently inaccurate scales; in others a mistake in recording the figures.

Valuable as is this check on stated weights, particularly in our overweight group, I feel that the greatest value of this table will prove to be our ability to classify the type of build with which we are dealing. One can state very definitely that the chest circumferences of the athlete are commonly larger than average and the abdomen smaller. The young adults in most cases have average or under average measurements. Men from 40 to 70 years of age usually have average or over average measurements and it will be seen that frequently the expansion is below normal and the abdomen in many cases excessive.

There are essentially five types of torso as related to the average—first, all measurements above average; second, all measurements average; third, all measurements below average; fourth, chest measurements over average with abdomen below average (athletic type); fifth, chest measurements small, abdomen large (pot bellied, obese, or senile type). These types are best illustrated by the following graph Fig. 1 in which the middle heavy line represents the average, the lines above indicating inches above average, and those below indicating inches below average. The first dot indicates the number of inches from average of chest at inspiration; the second, of chest at expiration; the third of abdomen.

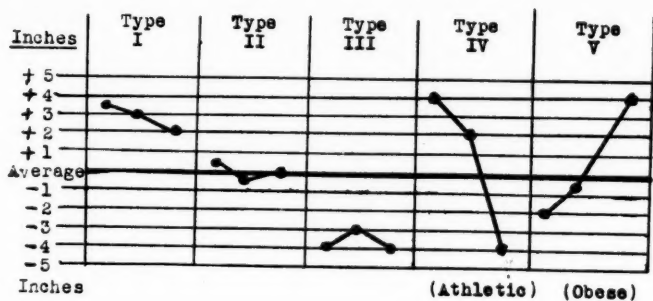


Fig. 1

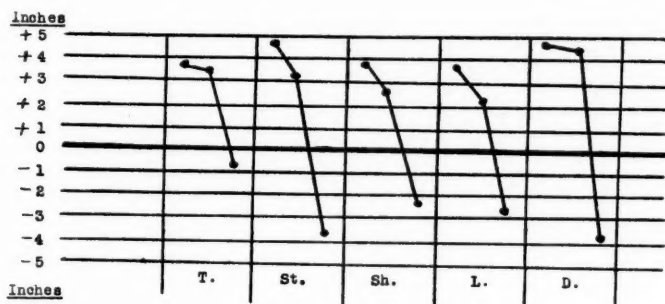


Fig. 2

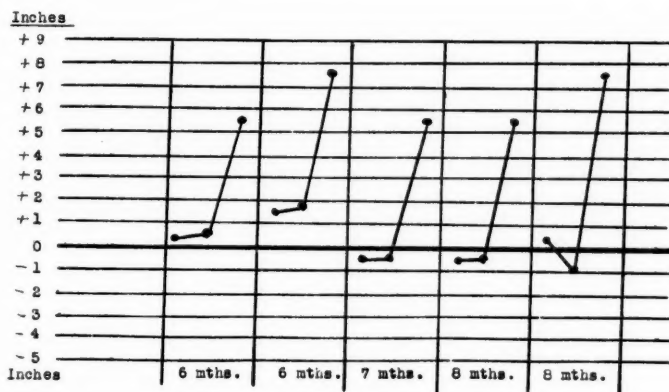


Fig. 3

These cases, Fig. 2, illustrate type 4 and are taken from the measurements of five internationally known boxers of the heavy weight class. Occasionally one may find an athlete who does not have this type of build, but it appears to be the usual type found in young athletes in training.

Figure 3 gives illustrations of women in the sixth to eighth months of pregnancy and illustrates the graph one would find with abdominal enlargement. (Type V.)

It would seem probable that the last is the least desirable of all the groups as it would include not only the cases of obesity but also abdominal enlargements from ascites, tumor, etc.

Interesting illustrations of this type are the following cases.

B. E. E.—Female—Age 64.

This applicant was declined by our Company but we understand that on the same examination, together with re-checking by two other Examiners, the applicant received a total of \$200,000 insurance.

Applicant, *within a month*, noted an increasing progressive enlargement of the abdomen, which was tapped, showing much bloody fluid. Shortly afterward she was operated upon and it was found that she had abdominal cancer. She died three months later.

Height 5' 8½", Weight 162 pounds. (3 pounds under average.)

		Theoretical for Height & Weight	Variation from Average
Chest at Insp.....	40	38.1	+1.9
Chest at Exp.....	37	34.5	+2.5
Abdomen	43	33.1	+9.9

Remarks:

This applicant appeared to be somewhat fat and at the time of examination showed a marked increase in size of abdomen, due undoubtedly to cancer and ascites.

At the time of the operation it was found that applicant had a disseminated carcinoma with marked involvement of

the omentum and much bloody fluid. It is apparent that an increase of 10 inches in abdominal girth over average, particularly in an individual of average weight, is strong evidence of pathology.

H. H.—Age 55.

Height and Weight 5' 7½", 178 lbs.—not weighed.

Chest Insp. _____ 42" = +2

Chest Exp. _____ 40" = +3.7

Abdomen _____ 42" = +6.3

Applicant was reported for a high blood pressure in 1926 and 1929. Three tests by our Examiner were entirely normal. The Examiner states that he was sure the applicant's heart and blood pressure were entirely normal and that the previous high records were due to the fact that they were taken after a heavy day's work. The first specimen showed .005% albumin, second .01%—and a few small hyaline casts.

We declined because of previous M. I. B. record, urinary findings, and a large abdomen. Applicant died within three months after a six months illness.

We wrote to our Examiner and ascertained that applicant died of cancer of the stomach. It appears possible that he may have had a metastatic carcinoma with bloody ascites, or the case may have been one of obesity.

O. E. D.—Age 52.

Height and Weight 5' 8¾", 202 lbs. Stated to have been weighed.

Chest at Insp. _____ 43½" = +1

Chest at Exp. _____ 41" = +2

Abdomen _____ 44" = +5.5

Applicant is 36 pounds overweight with a mortality of only 128%. We declined, however, because of trunk measurements with weight. Examiner described applicant as "obese, heavy set."

Remarks: This is apparently a case of obesity.

The value of this table in clinical medicine has yet to be determined, but steps are being taken at this time in one of the large clinics of our country to make a study of this subject. The value in insurance selection I believe to be evident and fortunately the method of employing the table

is so simple that any trained assistant can use it as a routine check.

In closing I desire to state that it was not until I had completed this study that I learned of a similar study of this problem by Dr. Weisse which was presented to this Association in 1913. His methods in arriving at the averages and the factors of increase are entirely different from those employed here. However, the results are very similar and I feel that one study corroborates the relative accuracy of the other. There is a distinct advantage, I believe, in incorporating the averages in a single table. In view of the fact that the study is based upon insurable ages 14 to 70 years, one must bear in mind the variation from average at the various decades as noted above, namely, that older men tend to have slightly larger measurements than the theoretical, and young men slightly smaller.

CHEST AND ABDOMINAL MEASUREMENTS AS
RELATED TO HEIGHT AND WEIGHT.

(STATISTICAL STUDY.)

JOHN S. THOMPSON, *Vice President and Mathematician, Mutual
Benefit Life Ins. Co.*

Much has been written concerning the relation between the height and weight of a human being. Attention was directed to this matter nearly a century ago by the Belgian Statistician, Quetelet, and numerous investigators since, and no doubt previously, have dealt with this interesting phase of anthropometry. Dr. Livi writing some years ago in *Italian Archives of Biology*, discusses various height-weight indices, a typical one being "the ratio of the individual's height to the side a cube containing a volume of water whose weight is equal to the weight of the individual." In a publication (No. 272) of the Carnegie Institute of Washington, Dr. C. R. Bardeen refers to an index of build obtained by dividing the weight by the cube of the height. Dr. Bardeen's comment is, in part, as follows: "The volumes of objects of the same shape but of different sizes vary as the cube of a given diameter through these objects. The volumes of the bodies of individuals of the same external form but of varying heights vary as the cube of the stature multiplied by a factor which is conditioned by the form of the body as π is conditioned by the form of a sphere. This factor, in the case of the relation of volume to stature, expresses the part of a space equal to the cube of the height occupied by the volume of the body. . . ." Dr. Charles B. Davenport of the same institution continues the discussion of the "Best Index of Build" in a recent number of the *Journal of the American Statistical Association* thus: "The naive procedure is to divide the weight by the height but this has the serious objection that while height is a linear dimension weight is really a volumetric dimension. Were men approximately cylinders of different height then the weight

divided by the height would give an approximate index of build but obviously the hypothesis is not in accordance with the facts. It has sometimes been urged that, since bodies of the same shape but of different size vary in volume as the cube of any diameter, the weight should be divided by the cube of the height in order to secure the index of build; but the hypothesis that men of different stature are of the same form is also not justified, for tall men have a prevailingly slender build and short men are on the average stockier....” This author proceeds to draw the conclusion that the best index of build is the weight divided by the square of the height. The diversity of indices discussed suggests that various investigators may have had different objects in mind for the use of such index, some intending to measure the degree of deviation from an average, others perhaps to establish an index of robustness or vitality.

Except for the article by Dr. Weisse before this Association, already mentioned by Dr. Clark, no reference appears to have been made, in any of the articles noted, to the possibility that the horizontal measurements of the body might have a place in the construction of an index which could be used in the estimation of weight.

Dr. Clark’s first conclusions were based on about one thousand cases selected at random from the current applications for insurance. These were so interesting as to justify the systematic study of a larger volume of data and pursuant to this plan the necessary details were drawn from approximately 10,000 applications recently approved, also selected at random. In each case the examiner had stated that the weight was by scale.

It was assumed that the weight might be expressed as

$$\text{Weight} = \text{Height} \times f(m)$$

where $f(m)$ denotes some function of one of the horizontal measurements or of the mean of two or more. Consideration was, therefore, given to the quotient

$$\text{Weight} \div \text{Height},$$

and an endeavor has been made to determine the law, empirical

or otherwise, expressed by $f(m)$. The quotient, Weight divided by Height, will be denoted by HW and called the "Height-Weight Index," although it is different from most indices heretofore discussed. In all the studies described here, m was taken as the sum of the three measurements,—Chest at Inspiration, Chest at Expiration, and Abdomen—the sum (S) being used for convenience instead of the mean. To secure the advantage of working with integers, fractional measurements of weight, height, etc. were taken to the nearest integer.

A group of average heights, 5'7" to 5'11", were first studied. In all groupings it was observed that the Height-Weight Index increased, with increase in S , at an increasing rate up to a point at which S was approximately equal to 105 or 110, after which it increased at a decreasing rate. This precluded the assumption that $f(m)$ was of the nature of

$$k \cdot S^p$$

where k would be a constant depending on the shape and specific gravity of the body and p would be between 1 and 2. For several groups p was found to be about 1.35 but the progression of HW was distinctly different from that of the unadjusted data.

The curve

$$HW = (S + qHW + r) (U - HW) (HW - L) + lS + mHW + n$$

of the third degree, having the contour just described, namely that HW increases with a uniform rate of increase in S , at an increasing rate over one portion of its course and subsequently at a decreasing rate was then fitted to the data. In this formula, which is, of course, quite empirical, U and L are, respectively, practical upper and lower limits to HW. The constants q , r , l , m and n were determined by the method of Least Squares.

The use of mean heights had the result of offering very meagre data in connection with the extreme values of HW, that is, under 25 and over 30. All the available data in connection with very low and very high values of S and HW were therefore analysed separately and are perhaps of sufficient interest to warrant tabulation here:

Thompson—Measurements and Weights 355

TABLE NO. 1.

S (Inches)	Number of Cases	Average HW (Total W ÷ Total H)
77	4	16.512
78	4	17.700
79	3	17.933
80	5	18.221
81	7	18.932
82	19	19.330
83	20	19.415
84	12	20.107
85	23	20.971
86	29	20.729
87	32	21.271
88	52	21.619
89	65	22.139
90	100	22.563
91	116	22.601
92	171	23.205
93	199	23.508
94	257	23.590
95	323	24.138

110	258	29.842
111	231	30.271
112	219	30.687
113	226	31.077
114	163	31.199
115	151	31.873
116	137	32.155
117	115	32.353
118	119	32.843
119	80	32.918
120	58	33.409
121	53	33.586
122	50	33.615
123	31	34.191
124	37	34.085
125	18	34.350
126	13	35.607
127	13	34.330
128	10	33.963
129	6	33.780
130	4	36.660
131	3	33.509
132	3	34.514
133	1	36.000
134	3	36.514
135	—	-----

The character of these values of HW, which proceed rather irregularly, will be better understood if the groups are consolidated thus:

TABLE NO. 2.

S	Number of cases	Average S	Average HW
77-82	42	80.52	18.621
83-87	116	85.35	20.641
88-92	504	90.57	22.841
113-117	792	114.69	31.626
118-122	360	119.54	33.168
123-127	112	124.46	34.363
128-133	26	129.35	34.357
130-134	14	131.71	35.456

The extreme groups are seen to be very small. However, due weight was given to these observations and with the adjustments of the preliminary series, which were considered necessary, values of HW, exemplified by the following specimen values, were arrived at which were considered a fair interpretation of the cases examined:

TABLE NO. 3.

S	HW
80	18.430
85	20.489
90	22.461
95	24.033
100	25.866
105	27.823
110	29.812
115	31.743
120	33.524
125	34.771
130	35.348

This may be used to construct the following table showing the average weight for various heights and various values of S.

TABLE NO. 4.

[illegible]

The "fit" of this table was tested by observing the deviation of the theoretical value as given in the table from each item in the original data. For example, according to the table the average weight of an individual whose height is 5' 10" and the sum of whose horizontal measurements is 110" is 174 pounds; if an individual with these linear measurements appeared in the original data with a weight of 170 pounds, the "error" from use of the table would be + 4 pounds whereas if the actual weight were 180 pounds, the "error" would be called — 6 pounds. To reduce the work involved in this process and at the same time preserve a reasonable distribution of the tested data among high, low and middle ages, the test was applied only to age groups 23—27, 38—42 and 53—57 inclusive. The results were:

TABLE NO. 5.

"Error" (1)	No. of Cases (2)	Per Cent of Total (3)	Total of "Errors" (4)	Per Cent of Total (5)
+ 11 lbs. or more	356	11.5%	5548	24.4%
+ 6 lbs. to + 10 lbs.	462	14.9	3616	15.9
+ 1 lb. to + 5 lbs.	676	21.8	1883	8.2
Total Positive	1494	48.2%	11047	48.5%
0	137	4.4	0	0
— 1 lb. — 5 lbs.	620	20.0%	1884	8.2%
— 6 lbs. — 10 lbs.	455	14.6	3601	15.8
— 11 lbs. or more	397	12.8	6230	21.5
Total Negative	1472	47.4%	11715	51.5%
Grand Total	3103	100.0%	668	

It is seen from column (3) that about 46% of the original data lie within 5 pounds on either side of the value shown in the table and about 76% within 10 pounds on either side of that average. The various positive and negative errors are well balanced, with the possible exception of the extreme groups in column (4). In general the group is, by nature, rather diffuse and the table of averages would be much more useful if the tendency were towards greater concentration about the mean.

Similar information in connection with each of the three age groups used in the test of fitness is as follows:

TABLE NO. 6

	Ages 23-27				Ages 38-42				Ages 53-57			
	No. of Cases (2)	Per-Cent (3)	Total "Errors" (4)	Per-Cent (5)	No. of Cases (6)	Per-Cent (7)	Total "Errors" (8)	Per-Cent (9)	No. of Cases (10)	Per-Cent (11)	Total "Errors" (12)	Per-Cent (13)
"Error" (1)												
+ 11 lbs. or more	158	10.0	2401	20.8	151	12.2	2412	26.3	47	16.3	735	35.7
+ 6 lbs. to + 10 lbs.	229	14.4	1816	15.8	172	13.9	1329	14.5	61	21.2	471	22.9
+ 1 lb. to 5 lbs.	340	21.5	890	7.7	277	22.5	815	8.9	59	20.5	178	8.6
Total Positive	727	45.9	+5107	+44.3	600	48.6	+4556	+49.7	167	58.0	+1384	+67.2
0	73	4.6	0		45	3.7	0		19	6.6	0	
- 1 lb. to - 5 lbs.	317	20.0	964	8.4	251	20.4	772	8.4	52	18.0	148	7.2
- 6 lbs. to - 10 lbs.	249	15.8	1987	17.2	175	14.2	1369	14.9	31	10.8	245	11.9
- 11 lbs. or more	217	13.7	3464	30.1	161	13.1	2485	27.0	19	6.6	281	13.7
Total Negative	783	49.5	-6415	-55.7	587	47.7	-4626	-50.3	102	35.4	-674	-32.8
Grand Total	1583		-1308		1232		- 70		288		+710	

The percentages tabulated in connection with Total Errors are the respective percentages of total errors regardless of sign.

The proportions of cases falling within 5 pounds on either side of the average in three age groups are respectively 46%, 47% and 45% and within 10 pounds on either side of the average, respectively, 76%, 75% and 77%, thus showing that the "dif-fuseness" of each sub-group is about the same as that of the whole group. In case of the low age group, however, the total error is negative and indicates that the average is understated by about one pound. On the other hand, the net error in the oldest age group is positive and indicates that the mean is over-stated by about $2\frac{1}{2}$ pounds.

It must be evident that the average represented by Table No. 4 is a very broad one, that is, that a fairly large percentage of the cases are separated by a substantial interval from the mean. Furthermore, the data at the extreme builds, that is, where the sum of the three circumferences is under 85 inches or over 125 inches are very meagre; in approximately 10,000 cases there were found 78 of the former and 56 of the latter. The deviations at such points will be more violent and the results in practice cor-respondingly less helpful.

One must also keep in mind that the weights in Table No. 4 are *averages* and being built up from various builds will not apply with consistency to extremes of build. For example, the weight of overweight individuals, that is, those of a given circumference who are less than the average height, will be regularly underestimated because the HW index is built up from those of average height. Again, being based on the facts con-tained in the applications, the results include the effect of cases in which the weights have been intentionally understated. Dr. Clark's table, however, has been built up on the assumption that HW increases at a uniform rate over the entire range of values of S met in practice. We recognize that this understates the average weights at the lower values of S and overstates the average weights at the higher values of S but it seems to have

Discussion—Measurements and Weights 361

evolved a table, which is of practical value in estimating the average of applicants who are under or over the average weight for their respective measurements.

Similar studies are under way in connection with the Chest at Expiration, alone, and Abdominal Girth, alone, and with the sum of these two measurements, rather than of the three usual measurements. If the results throw any further light on the possibility of estimating weights from linear dimensions, they will, of course, be offered at the earliest opportunity.

DR. MUHLBERG—This is really a very interesting paper and shows the necessity of re-checks.

No other of our members is better able to discuss this paper than Dr. Faneuil S. Weisse of the Mutual Life of New York and we will ask him to give us his opinions.

DR. WEISSE—Gentlemen, Dr. Muhlberg used a word that I must object to in reference to this paper, that is, "discussion." There isn't anything to discuss, because it is cold facts, and you can't get away from it.

I have taken a great deal of interest in studying this paper, because Dr. Clark and I arrived at approximately the same conclusions by entirely different routes and by approaching the subject in two entirely different ways.

I was greatly interested in this very clever paper which Dr. Clark has given us, as I read an article before this Association with reference to this same subject in 1912.

A comparison of the average expiration, inspiration and abdominal measurements reached by Dr. Clark and by me for heights 5'3" to 6', with the same standard weights, vary in but two instances more than $\frac{1}{2}$ inch, as you will see by a study of the comparison. Dr. Clark, however, went further than I did and worked out a table which anybody can have on his desk and readily determine at a glance whether or not the measurements given for an applicant are approximately correct. This is a great advance over my idea in that the figures which I gave the Association required a mathematical calculation every time

they were used, to determine what the approximate measurement for a given height and weight should be.

COMPARISON OF MEASUREMENTS OF CHEST AND ABDOMEN WITH Table 1 Weisse Chart Based on 3035 Male Adults
Assoc. of Life Ins. Med. Directors Vol. 1913-1915—Page 129.

	<i>Inspiration</i>			<i>Expiration</i>			<i>Abdomen</i>		
	Weisse	Clark	Diff.	Weisse	Clark	Diff.	Weisse	Clark	Diff.
5.3 -138	35.85	— 36.3	.45	32.43	— 32.9	.47	31.55	— 31.0	.55
5.4 -141	36.06	— 36.5	.45	32.68	— 33.0	.22	31.39	— 31.1	.29
5.5 -148	36.45	— 37.2	.75	33.16	— 33.6	.44	32.07	— 31.9	.17
5.6 -149	36.65	— 36.9	.25	33.17	— 33.3	.13	32.09	— 31.6	.49
5.7 -154	36.92	— 37.2	.28	33.43	— 33.6	.17	32.38	— 31.9	.48
5.8 -158	37.12	— 37.5	.38	33.49	— 33.9	.41	32.36	— 32.3	.06
5.9 -163	37.64	— 37.9	.26	33.93	— 34.3	.37	32.97	— 32.8	.17
5.10-164	37.74	— 37.9	.16	33.84	— 34.3	.46	32.63	— 32.8	.17
5.11-169	37.89	— 37.9	.01	34.10	— 34.4	.30	33.01	— 33.0	.01
6.0 -178	38.84	— 38.8	.04	34.92	— 35.2	.28	33.86	— 34.0	.14

We are greatly indebted to Dr. Clark for a very favorable addition to our working tools and I congratulate him on his production.

DR. MUHLBERG—The next speaker has been an active member and willing worker with us for a number of years. I understand he has been using these tables in his office for a number of months and the discussion will be continued by Dr. Charles B. Piper of the Guardian Life.

DR. PIPER—Mr. President and Gentlemen: We represent a handful of the faithful. I think we demonstrate ability to absorb punishment and should be congratulated on our staying powers. I think it is eminently fitting that the discussers of this paper should be one Weisse and the other foolish.

It occurred to me that possibly an impression had gained circulation that I was a friend of Dr. Clark's and I assumed that in excess gratitude he immediately rushed to my office and provided me with a copy of his table to employ. We have been using that table and I only regret that we have had only a few months in which to try it. It is a very easily handled table and it has been instrumental in affording us a great deal of help, and I believe, has meant a distinct saving in dollars and cents. We

Discussion—Measurements and Weights 363

have written back to a number of examiners in order to bring into apparent relationship abdominal and chest measurements and the weight given, and we have in each case insisted that the examiner should not only re-weigh the applicant if possible, but that he should give us new abdominal and chest measurements. Frequently what appeared to be a disparity has been corrected, a very obvious misstatement of figures in the original report. We have not written back when the apparent deviation would not materially affect our acceptance of the risk. Scarcity of time was responsible for this, but when it seemed to us probable that it would bring the risk into a ratable class, then we have insisted upon exact measurements, sometimes, as Dr. Clark suggested to me, writing a second time and urging the possibility of the scale being erroneous. The examiner, being given that happy opportunity of executing a retrograde movement, was able eventually to write and tell us that his original weight had been misstated, in the largest case by a matter of 47 pounds. It is illuminating to check up the work of examiners and have your confidence in certain trusted examiners shaken, and to come to view subsequent estimates of height with a great deal of reluctance.

Now to terminate with just a few figures. Having had the table since about the first of July, we endeavored, with a comparatively small class of risks, to get some idea of the accuracy with which our measurements of applicants examined at the Home Office would check with Dr. Clark's table. Over 300 cases were examined at the Home Office, and we took particular care to measure the chest and the abdomen with a steel tape and to take the height with the standard on a Howe scale and to weigh each applicant with a very closely tested scale. We found that there were 112 cases in which our measurements in the Home Office showed a weight which was in *excess* of the weight given in Dr. Clark's table. The average deviation was 7.81 pounds per applicant. The largest individual deviation was plus 24 pounds, that is, our weight was 24 pounds greater than the table of Dr. Clark. Regrettably, I had failed to ask our examiners to comment upon the apparent cause of the disparity in each abnormal deviation

from the table of Dr. Clark. I am unable to say whether it was due to a short neck, to large limbs or to whatever cause.

We had 174 cases in which there was a weight which was *less* than Dr. Clark's table showed, a deviation of 8.6 pounds. In other words there was a greater number of cases in which we found that our weight was under the weights given in his table. Now I believe that the explanation of that may be this: We are taking the measurements over the bare skin. It is an individual peculiarity with two of the Home Office examiners that they draw the tape very tightly. I made an estimation on all these cases, over 300, by adding $1\frac{1}{2}$ inches to the combined total, of two chest and one abdominal measurements. You take the abdominal, the chest after expiration and after inspiration, and if the total is 130 you read across to the height and find your given weight. I added arbitrarily $1\frac{1}{2}$ inches to the total. With the tape drawn tightly we found the deviation was 2.4 pounds per applicant in three hundred and one applicants examined at the Home Office. In percentage it is almost the same, 2.6 per cent. deviation from Dr. Clark's table. I think that is a very close approximation and I only regret, as I say, that I did not have the opportunity of extending my observation over a period longer than that of three months.

Again, Dr. Clark in taking his 9,000 cases took field examined cases in which they were weighed by the field examiner. Now there might possibly be a certain degree of inaccuracy in the scales used. It is quite possible, and, I think, altogether likely that there was a real error in the height. It is human nature for each man to compliment himself on his height. I have invariably found that a man who states that his height is 5 feet $9\frac{1}{2}$ inches is three-quarters or one inch less especially after middle age. We compliment ourselves upon those things. I wish very much indeed—and it might be possible—for different offices to report to Dr. Clark the result of Home Office examinations, the measurements being taken very accurately, the scales being carefully adjusted, and to see then whether there might be some revision of the table. I feel quite assured in our office at least

Discussion—Measurements and Weights 365

that we would find that it would make an almost perfect approximation of our figures with the figures of Dr. Clark. It is a splendid table, and very easily used.

In conclusion I can only say as the bibulous friend of Irvin Cobb said: "Have you ever had delirium tremens?" and on being answered in the negative, said: "If you have never had delirium tremens, you have never been nowhere and you have never seen nothing." If you have not used this table, you have seen nothing in estimating height and weight.

DR. MUHLBERG—Is there any further discussion? I will call on either Dr. Charles P. Clark or Mr. John S. Thompson.

DR. CLARK—May I first finish my discussion here. I want to thank Dr. Piper for doing as I asked. Let me suggest to Dr. Piper or rather ask Dr. Piper, "What do you suppose is the average age of the clerks examined, because that, as you see here, will make a material difference." If a majority of them are under 35 years of age, it will show just what you found, that you will under-estimate more than you will over-estimate. If we took all the men that were here in the Medical Directors' Association and tested them out, I should say that the average age would be well over 50, and then we would find something such as we have here, that we over-estimate on the average, and we would have very few that would be under-estimated. (To Dr. Piper) Did you have the average age of the Home Office clerks?

DR. PIPER—These were insurance applicants examined at the Home Office, Dr. Clark, and I have endeavored to see whether it might be possible to put them in age groups, but they seem to be very evenly distributed. The youngest age that I have on this table is age 22 and it goes up to 56.

DR. CLARK—I thought they were Home Office clerks. I believe the average age of applicants for most of the companies will run somewhere between 35 and 40, and if I am not mistaken, I think Mr. Thompson said the New York Life found theirs to be 37 or 38.

The fact that you did measure these over the skin and that your height was taken more accurately might account for the

difference. However, as to using the scale as it is, you don't realize how much work it is to make a scale of this kind. I would hate to hold off until we do it all over again, because it is a big job. I am willing to let the scale go as it is; I don't know if Dr. Weisse is or not.

DR. WEISSE—I am.

DR. MUHLBERG—Mr. Thompson, won't you come forward.

MR. THOMPSON—It would be very inconsiderate of me to take up your time at this late hour in the formal presentation of data which are already in your hands. I should, however, like to emphasize the fact that the table giving the weights for various builds is, as nearly as we could make it, an average of the approximately 9,400 cases entering into the investigation. From tables which are given you will see that about 50% of the original data fell within five pounds on either side of the average and that about 75% fell within ten pounds on either side of the average. Dr. Clark's method of constructing his table has the result of giving weights at the higher girth measurements, which are in excess of mine, and at the lower girth measurements, which are less than mine. A practical consequence of the use of such a table is that attention is drawn to the understatement of the weight of overweights at the larger girth measurements, and to the understatement of underweights at the lower girth measurements. Such a table for this reason has a practical value which is not shown by an exact analysis of the original data.

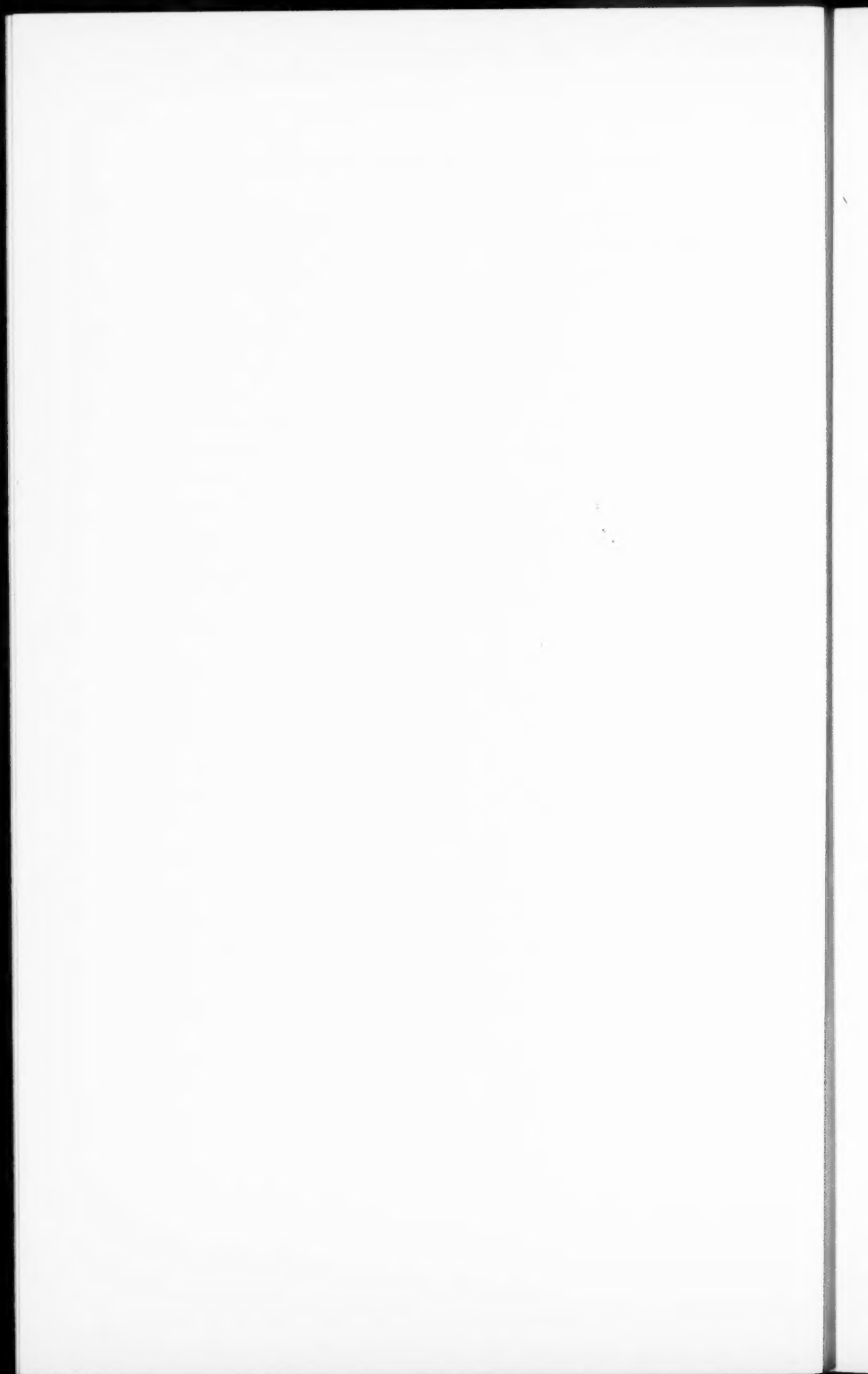
Dr. Piper has referred to the desirability of having tables based upon more exact measurements. This would be desirable and I hope that, in time, a more accurate group of statistics may be developed, but I believe that the groups are so diffuse that no matter how accurate the measurements are, a large fraction of the data will always lie so far from the average that we should hesitate before making further inquiries in connection with deviations from the average weight unless such deviations are fairly large, say over fifteen pounds or over 10% of the average weight.

It may be well to add that when the average weights which were used in constructing the table appearing in my memoran-

Discussion—Measurements and Weights 367

dum, are charted, the graph is concave to the build-axis in case of the higher girth measurements and convex to that axis in case of the lower measurements. In other words, the actual curve of averages is shaped like an elongated S. The reason for this is obvious, namely that for a given height and for the larger girth measurements we are constantly eliminating those of excess weight and for the small measurements we eliminate those with an unusual degree of underweight. Dr. Clark in constructing his table assumed that the graph could be regarded as a straight line whose direction was determined by the central portion of the graph based upon the average of all the data. This gives rise to the situation noted in the first paragraph and is, in fact, what gives Dr. Clark's table its practical value.

I appreciate very much the opportunity of coming before you and of having these few observations printed in your Proceedings.



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375

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385

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Cumulative Index

TO THE PROCEEDINGS OF

THE ASSOCIATION OF LIFE INSURANCE MEDICAL DIRECTORS OF AMERICA

1920-1929

	Volume	Page
Abnormal Pulse, Its Detection, Recognition and Evaluation. Gordon Wilson	xv	192
Discussion—Cook, Rogers, Old, Cragin.....		218
Acetone. The Determination of Acetone in the Urine and Its Significance in Life Insurance Examinations. Jeanette Allen Behre and Wm. Muhlberg	xvi	167
Discussion—Benedict, Cort, Brown		184-195
Actuarial Society, Joint Meeting.....	vi	98
Actuarial Society of America.....	xv	53
Actuaries:		
Relationship with Actuary. R. Henderson	xv	35
Address. W. W. Beckett.....	xiii, 196; xiv, 6	
Address of the President:		
T. H. Rockwell	vi	4
F. S. Weisse	vii	6
A. S. Knight	viii	6
T. F. McMahon	ix	9
F. L. Grosvenor	x	6
W. R. Ward	xi	16
F. S. Whitney	xii	17
A. B. Hobbs	xiii	12
W. W. Beckett	xiv	6
R. M. Daley	xv	12
J. A. Patton	xvi	11

	Volume	Page
Address of Welcome:		
C. A. Peabody	VII	4
F. H. Ecker	VIII	5
D. P. Kingsley	IX	6
J. R. Hardin	XI	5
E. I. Low	XII	4
J. C. McCall	XIII	76
F. L. Jones	XV	9
Advantages of Rate of Excretion Over Concentra- tion as the Clinical Criterion in Proteinuria and Glycosuria. William G. Exton and Anton R. Rose		
	XVI	143
Discussion—Benedict, Cort, Brown, McCrud- den, Exton	XVI	182-198
Age Incidence of Scapular Types, Its Possible Rela- tion to Longevity. W. W. Graves		
	x	60
Discussion—Dublin, Hoffman, Patton and Rogers	x	60
Albumin in the Urine, a Simple and Rapid Test for. W. G. Exton		
	VIII, 188; IX,	263
Discussion—Blakely, Pauli, Bradshaw	IX	263
Albuminuria, A Visual Guide to. H. W. Cook	IX	277
Albuminuria and Casts. E. F. Russell	VIII	270
Discussion—Daley, Dwight, Symonds	VIII	270
Amendment Regarding Payment of Dues as Pre- scribed in Article 1, Section B, of the By-Laws		
	XVI	10
Amendment, Report on	XIII	37
Amendment to Article III of the Constitution	x	39
Amendment to Article VIII of the Constitution	x	38
Amendment to Article XII of the Constitution	VIII	61
Amendment to By-Laws	VI	9
American Heart Association	XVI	31
American Life Convention, Delegates	XVI	4
Amiral, Hiram H. Reaction of the Diastolic Pres- sure to the Cardio-Respiratory Test	XIV	219

Cumulative Index

393

	Volume	Page
Anderson, H. B. Focal Infections in Relation to Life Insurance	IX	144
Anthropometrical Supplement to Report of Committee on Dreyer's Work. L. I. Dublin	x	162
Anthropometry.		
Chest and Abdominal Measurements as Related to Height and Weight		
(Statistical Study) John S. Thompson	xvi	352
Chest and Abdominal Measurements as Related to Height and Weight, with Presentation of Tables of Averages. Charles P. Clark	xvi	341
Apical Cardiac Murmurs. A. E. Johann	xiv	155
Discussion—Wilson and Wenstrand	xiv	168
Apparatus for Obtaining the Specific Gravity of Minute Quantities of Urine. W. G. Exton	vii	289
Appendicitis, Some Effects of. R. L. Rowley	vii	280
Discussion—Gage, Hutchinson, Weisse, Dwight, Beckett, Snow	vii	280
Mortality rates	xvi	254
Archibald, T. D. Tuberculous Disease of Bone and Joints as Affecting Insurability	xiv	310
Assessment, Report of Committee on. C. L. Christiernin	xi	27
Auditing Committee	xvi	9
Aviation and Life Insurance. L. G. Sykes and W. B. Smith	xiv	74
Discussion—Bartlett, Smith and McCulloch	xiv	141
Bailey, W. C. Report on Diastolic Blood Pressure	xi	143
Baker, Henry A.		
Discussion of Paper on Women as Life Insurance Risks	xvi	336
Non-Medical Factors Affecting Mortality	xii	211
Battle, J. T. J.		
Tuberculosis from an Insurance Viewpoint	xv	266

	Volume	Page
Discussion—Ordway and Rollins	xv	269
Introductory remarks	xv	264
Beckett, W. W.:		
Address by	xiii, 196; xiv, 6	
Disability Insurance	xiv	17
Tuberculosis in Its Relation to Life Insurance	ix	115
Behre, Jeanette Allen, and Wm. Muhlberg:		
The Determination of Acetone in the Urine and Its Significance in Life Insurance Examina- tions	xvi	167
Discussion—Benedict, Cort, Brown		184-195
Benedict, S. R. Metropolitan Laboratories	x	111
Discussion of Papers	xvi	182-185
Bibliographies.		
Functional Tests of the Circulation	xvi	111-113
Medical Aspects of Intestinal Diseases	xvi	252
Peptic Ulcer and Biliary Disease	xvi	293
Biliary Tract, Disease.		
Bibliography	xvi	293
Biliary Colic. Mortality Study of Impaired Lives. Oscar H. Rogers and Arthur Hunter. No. 7.		
(a) Renal Colic (b) Biliary Colic	xv	412
Mortality Tables	xvi	254
Results of Treatment	xvi	286
Billing, Albert W.	xv	6
Blatherwick, Norman R. and Otto Folin. Blood Sugar Curves After the Ingestion of 50 grams of Glucose.		
Discussion—Benedict, Cort, McCrudden	xvi	155-158
Discussion—Benedict, Cort, McCrudden	xvi	183-197
Blood, Circulation. Functional Tests of the Circu- lation. L. F. MacKenzie, P. V. Wells, E. G.		
Dewis, L. S. Ylvisaker	xvi	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	xvi	114-135

Cumulative Index

395

	Volume	Page
Blood Pressure, as Affected by Sex, Weight, Climate, Altitude, Latitude, or by Abstinence from Alcoholic Beverages. A. Hunter and O. H. Rogers	VI	92
Blood Pressure, Further Report of the Diagnostic Value of the Systolic and Diastolic Blood Pressure. J. W. Fisher, Chairman of Committee on Blood Pressure	VII	22
Discussion—Rogers, Root, Rockwell, Toulmin, Wells	VII	22
Blood Pressure of Healthy Men and Women. B. Symonds	IX	22
Discussion—MacKenzie, Ward and Fisher.....	IX	22
Blood Pressure, Some Practical Observation on the Taking of. G. E. Crawford	XI	38
Blood, Sugar.		
Blood Sugar Curves After the Ingestion of 50 Grams of Glucose. Otto Folin, and Norman R. Blatherwick	XVI	155
Turbidity Micro Method for Blood Sugar. Anton R. Rose, F. Schattner, and William D. Exton.....	XVI	178
Bradshaw, William M., Focal Infection in Relation to Life Insurance	VII	222
Glycosuria. Discussion	XV	122
Bradshaw, William M. and Frank H. Carber. The Clinical Significance of Intermittent Glycosuria from the Viewpoint of Insurance Medicine	XV	66
Brathwaite, Frederick G.		
Cardiography and Oscillometry in Life Insurance Selection	XVI	19
Discussion—Old, Cragin	XVI	31-35
Glycosuria—Charts	XV	100-107
British Royal Air Force Breath-Holding Test.....	XVI	48
Brown, Chester T. Female Risks Which Have Undergone Intra-Abdominal Operations Upon the		

	Volume	Page
Pelvic Genital Organs, the Selection and Mortality Experience of	VI	31
Brown, Frederick R. A Study of Urinary Analyses	XIV	343
Discussion of Papers	XVI	192
Brown, Lawrason, The Value of the X-ray in the Diagnosis of Pulmonary Tuberculosis.....	VII	343
Cancer. Cancer of the Stomach	XVI	241
Cancer. Relation Between Overweight and Cancer—a Preliminary Examination of Evidence from Insurance Statistics. Louis I. Dublin	XV	402
Discussion—Muhlberg	XV	408
Cancers, Address on. H. R. Gaylord	VIII	231
Discussion—Cook and Rowley	VIII	231
Carber, Frank. The Present Status of Goiter	XI	270
Carber, Frank H. and William M. Bradshaw. The Clinical Significance of Intermittent Glycosuria from the Viewpoint of Insurance Medicine.....	XV	66
Carcinoma, The Chances of Death from, J. Ewing.....	XIII	50
Discussion—Knight and Toulmin	XIII	61
Cardiac Dyspnea. Summary of Professor F. R. Fraser's Goulstonian Lectures	XVI	93
Cardiac Functional Tests, Introductory Remarks on. E. W. Dwight	IX	203
Cardiography and Oscillometry in Life Insurance Selection. Frederick G. Brathwaite	XVI	19
Discussion—Old, Cragin	XVI	31-35
Cardio-Respiratory Test	XVI	123
Cardio-Respiratory Test: Analysis of Experience for the Past Five Years. H. M. Frost.....	XIV	183
Discussion—Grosvenor, Patton and Wilson.....	XIV	282
Cardio-Respiratory Test, Report on. H. M. Frost	XII	62
Discussion—Dwight and Rogers	XII	67

Cumulative Index

397

	Volume	Page
Cardio-Vascular Degeneration. H. W. Cook.....	XIV	250
Introduction	XIV	247
Discussion—Grosvenor, Patton and Wilson.....	XIV	282
Cardio-Vascular Reaction to Abnormal Variations of Intrathoracic Pressure, A Study of, H. M. Frost	IX	207
Discussion—Exton, Symonds, Pauli, Wilson, Porter, Archibald, Rogers	IX	207
Chadwick, Henry D. The Diagnosis and Prognosis of Juvenile Tuberculosis	XIV	327
Chapin, Frank W.	XVI	5
Chapman, John P. Standardization of Medical Ex- amination Blanks	VIII	169
Chest and Abdominal Measurements as Related to Height and Weight (Statistical Study) John S. Thompson.....	XVI	352
Discussion—Weisse, Piper	XVI	361-362
Chest and Abdominal Measurements as Related to Height and Weight, with Presentation of Tables of Averages. Charles P. Clark	XVI	341
Discussion—Weisse, Piper	XVI	361-362
Circulation.		
Functional Tests of the Circulation. L. F. Mac- Kenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	XVI	36
Bibliography	XVI	111-113
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	XVI	114-135
Clark, Charles P. Chest and Abdominal Measure- ments as Related to Height and Weight with Presentation of Tables of Averages	XVI	341
Discussion—Weisse, Piper	XVI	361-362
Clark, Charles P. Demonstration of Urinary Tests	XI	250

	Volume	Page
Clinical Significance of Intermittent Glycosuria from the Viewpoint of Insurance Medicine. William M. Bradshaw and Frank H. Carber	xv	66
Introductory Remarks by Carber	xv	63
Cole, Lewis Gregory, The Roentgenological Selection of Latent or Non-Clinical Pulmonary Tuberculosis Lesions	vii	354
Colitis	xvi	246
Colorimeter, The. D. B. Cragin	xiii	194
Committee—Auditing	xvi	9
Committee, Joint (Actuarial and Medical) on the Influence of Family History on Mortality.....	viii	56
Committee—M. I. B.	xvi	9
Committee, Nominating	xvi	8
Committee on Advisability of Informing Hospitals and Physicians on Cause of Death of Insured Persons Previously Treated by Them	vii, 243; viii, 59	
Committee on Blood Pressure	viii, 38; ix, 59; x, 35	
Committee on Dreyer Measurements in Relation to Life Insurance Underwriting Practice. W. Muhlberg	xi	28
Discussion—Pauli and Dwight	xi	28
Committee on Dreyer Measurements in Relation to Life Insurance Underwriting Practice, 1925, Report of, L. I. Dublin	xii	204
Committee on Medical Examination Blanks	x	36
Committee on Public Health	viii, 34; ix, 19; x, 36	
Committee on Revision of the Constitution and By-Laws	x	38
Committee on the Standardization of the Medical Blank, Report of	ix	21
Committee on the Work of Dreyer in Relation to Life Insurance, Report of	ix	19

Cumulative Index

399

	Volume	Page
Committee on Urinary Impairments, Report of, J. A. Patton	XI, 244; XII, 115; XIII, 34; XIV, 12	
Discussion—Cook, Rockwell, Bradshaw, Rogers and Knight	XI	253
Discussion—Dwight, Cragin and Muhlberg	XII	116
Committee, Report of Nomination	IX	17
Committee Reports	XIII, 32; XIV, 10	
Committee to Draft Resolution in Regard to Dr. Rogers	XVI	305
Committee to Make a Survey of the Medical Ex- aminer Situation	X	43
Committee to Review Questions on Medical Blanks, Report of, H. Toulmin	XII	18
Committee to Review the Constitution and By-Laws, Appointment of	IX	201
Committee to Study and Report on Advisability of Educating and Listing Examiners	XV	190
Committee to Study Dreyer's Work in Relation to Life Insurance	VIII, 309; X, 147	
Committee to Study the Question of the Standardi- zation of Medical Blanks	VIII	309
Companies and Their Representatives. VI, 264; VII, 369; VIII, 322; IX, 292; X, 297; XI, 359; XII, 307; XIII, 311; XIV, 460; XV, 442; XVI, 380		
Conservation Work, Based on Annual Urinalysis and Policyholder's Statement of Health. W. Muhlberg	VI	12
Discussion—Willard, Wells, McCulloch, Jaquith	VI	12
Cook, Henry H. Cardio-Vascular Degeneration	XIV	250
Cook, Henry W. Discussion of Paper on Func- tional Tests of the Circulation	XVI	135
Cook, Henry Wireman. A Visual Guide to Albuminuria	IX	277

	Volume	Page
Cragin, D. B.		
Discussion of Paper on Surgery of Digestive Organs	xvi	225
The Colorimeter	xiii	194
Craig, James D. Disability Benefit in Life Policies..	xi	294
Crawford, G. E. Some Practical Observations on the Taking of Blood Pressure	xi	38
Cullen, George. Discussion of Paper on Women as Life Insurance Risks	xvi	332
Daley, Robert M.		
Diabetes	xv	76
Relationships of Medical Directors	xv	12
Delegates from American Life Convention	xvi	4
Demonstration of Urinary Tests. C. P. Clark and F. B. Kingsbury	xi	250
Discussion—Cook, Rockwell, Bradshaw, Benedict and Folin	xi	250
Determination of Acetone in the Urine and Its Sig- nificance in Life Insurance Examinations. Jean- nette Allen Behre and William Muhlberg.....	xvi	167
Discussion—Benedict, Cort, Brown	xvi	184-195
Dewis, E. G.		
Functional Tests of the Circulation. L. F. Mac- Kenzie, P. V. Wells, E. G. Dewis, and L. S. Ylvisaker	xvi	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	xvi	114-135
Diabetes. Robert M Daley	xv	76
Diabetes.		
The Determination of Acetone in the Urine and Its Significance in Life Insurance Examina- tions. Jeanette Allen Behre and William		

Cumulative Index

401

	Volume	Page
Muhlberg	XVI	167
Discussion—Benedict, Cort, Brown	XVI	184-195
Diabetes and Life Insurance. E. P. Joslin	VIII	87
Discussion—Muhlberg, Extton, Balch and Rogers	VIII	87
Diagnosis and Prognosis of Juvenile Tuberculosis. H. D. Chadwick	XIV	327
Discussion—Knight, Bailey and Wilson.....	XIV	335
Diastolic Blood Pressure. W. C. Bailey.....	XI	143
Digestive Tract.		
Disability and Digestive Disorders. H. Dingman	XVI	285
Operative Mortality	XVI	214
Surgery of the Digestive Organs. J. M. T. Finney	XVI	203-218
(See also: Dyspepsia, Peptic Ulcer Gastro- Intestinal Tract.)		
Dillard, Henry K. Standardization of Medical Ex- amination Blanks	VIII	170
Dingman, H. W.		
Disability Insurance	XII	263
The Doctor and Insurance	XIV	295
Disability and Digestive Disorders	XVI	285
Discussion—Wilson, Harnden, Sykes, Muhl- berg, Dingman	XVI	294-304
Directions for Applying Functional Tests	XVI	102
Disability and Digestive Disorders. H. Dingman.....	XVI	285
Discussion—Wilson, Harnden, Sykes, Muhl- berg, Dingman	XVI	294-304
Disability and Mortality in Arrested Tubercular Cases. E. S. MacSweeney	XII	142
Discussion—Howk, Wilson, Sykes, Hobbs and Battle	XII	187

	Volume	Page
Disability Benefit in Life Policies. J. D. Craig _____	XI	294
Discussion—Beckett, Grosvenor, Root, and Snow _____	XI	294
Disability Benefits:		
Disability Benefits. George P. Paul _____	xv	354
Discussion—Olsen, Wells, Wehner _____	xv	369-401
Total and Permanent Disability Benefits for Women. John Ferguson _____	xv	302
Disability Benefits, Selection from the Underwriting Standpoint. A. D. Reiley _____	xiii	287
Disability Claims. Tables _____	xvi	296
Disability Insurance. W. W. Beckett _____	xiv	17
Discussion—Van Dervoort, Root, Rockwell, Frost and Rogers _____	xiv	34
Disability Insurance H. W. Dingman _____	xii	263
Discussion—Foshay and Sykes _____	xii	284
Diverticulitis of the Colon, Acute. J. F. Erdmann _____	xii	96
Doctor and Insurance. H. W. Dingman _____	xiv	295
Dreyer Measurements in Relation to Life Insurance Underwriting Practice, Committee on, W. Muhl- berg _____	xi	28
Dreyer Measurements in Relation to Life Insurance Underwriting Practice, 1925 Report of the Committee on, L. I. Dublin _____	xii	204
Dreyer, the Work of, in Relation to Life Insurance. L. I. Dublin _____	viii	202
Discussion—Fisk and Symonds _____	viii	202
Dublin, Louis I. Anthropometrical Supplement to Report of Com- mittee on Dreyer's Work _____	x	162
1925 Report of the Committee on Dreyer Meas- urements in Relation to Life Insurance Under- writing Practice _____	xii	204
The Work of Dreyer in Relation to Life Insurance _____	viii	202

Cumulative Index

403

	Volume	Page
Dublin, Louis I. The Relation Between Overweight and Cancer — a Preliminary Examination of Evidence from Insurance Statistics	xv	402
Discussion—Muhlberg	xv	408
Dublin, Louis I. and Augustus S. Knight. Mortality, Morbidity and Working Capacity of Tuberculosis Patients After Discharge from the Metropolitan Life Insurance Company Sanatorium Between 1914 and 1927	xv	247
Discussion—Ordway, Rollins, Fellows	xv	269-281
Dunham, Kennon. X-Ray Chest Examinations, A Practical and Valuable Method for the Application of.	ix	132
Duodenal Ulcers. (See Peptic Ulcers.)		
Dwight, E. W. Cardiac Functional Tests. (Introduction to Dr. Harold M. Frost's Paper.)	ix	203
Selection of Risks by Methods Other Than the Numerical Rating	vii	184
Dyspepsia	xvi	237
Analysis of 1,650 Cases with Gastric or Dyspeptic Symptoms. Blackford and Dwyer	xvi	240
Eakins, O. M. Medical Directors	x	128
"Mistakes"	xiv	46
Numerical Ratings in the Selection of Risks.....	vii	194
Effect on Mortality of a History of Tuberculosis. T. H. Rockwell and R. Henderson	xiv	396
Discussion—Rogers, Scadding and Weisse.....	xiv	409
Election of Honorary Members	x	42
Election of New Members	vi, 3; vii, 3; viii, 3; ix, 3; x, 3; xi, 3; xii, 3; xiii, 3; xiv, 3; xv, 3; xvi, 3	

	Volume	Page
Election of Officers.....vi, 50; vii, 165; viii, 32; ix, 200; x, 34; xi, 142; xii, 114; xiii, 138; xiv, 325; xv, 8; xvi, 201		
Electrocardiograph	xvi	17, 18
Cardiography and Oscillometry in Life Insurance		
Selection.: Frederick G. Brathwaite	xvi	19
Reasons for Using in Selection for Life Insurance	xvi	22
Emeritus Membership, Dr. T. F. McMahon	xv	4
Epilepsy in Relation to Life Insurance. J. Ferguson	ix	87
Discussion—Jenney, English, Fisher	ix	87
Erdmann, John F. Acute Diverticulitis of the Colon	xii	96
Euscopy, W. G. Exton	vii	290
Ewing, James. The Chances of Death from Carcinoma	xiii	50
Executive Council, 1928	xv	7
1928	xv	8
1929	xvi	v
Minutes of Meetings of May 16, 1929 and October 23, 1929 Adopted as Read by the Secretary..	xvi	4
Exercise Test	xvi	51
Comparison of Observed and Calculated Exercise Required to Delay Systolic Return Two Minutes	xvi	59
Standard Number of Ascents. Males	xvi	102
Standard Number of Ascents, Female	xvi	102
Experience of Certain Border-Line Classification.		
F. L. Grosvenor	x	6
Exton, William G.		
Albumin in the Urine, a Simple and Rapid Test for	ix	263
Euscopy	vii	290
Preliminary Communication of a Method for Estimating the Significance of Glycosuria	ix	258
Presentation of Apparatus, etc.	vii	289
Prudential Longevity Service	x	244

Cumulative Index

405

	Volume	Page
Quantitative Microscopic Urinalysis	XV	146
Discussion—Patton and Clark	XV	182
The Scopometer	XIII	109
Some Incidentals of Urinalysis	VII	292
A Simple and Rapid Test for Albumin	VIII	188
A Simple and Rapid Quantitative Test for Sugar in Urine	XIV	441
The Photo-Electric Scopometer	XVI	141
Discussion—Benedict, Cort, Brown, Exton....	XVI	182-199
Exton, William G. and Anton R. Rose. The Ad- vantages of Rate of Excretion Over Concentra- tion as the Clinical Criterion in Proteinuria and Glycosuria	XVI	143
Discussion—Benedict, Cort, Brown, McCrud- den, Exton	XVI	182-198
Exton, William G., Anton R. Rose and F. Schattner. Turbidity Micro for Blood Sugar	XVI	178
Discussion—Benedict, Cort, Brown	XVI	185-195
Exton, William G., A. Rose and P. V. Wells. A Simple and Rapid Quantitative Test for Sugar in Urine	XIV	441
Fellows, Haynes H. Discussion of Paper on Med- ical Aspects of Certain Gastro-Intestinal Tract Diseases	XVI	263
Female Risks Which Have Undergone Intra-abdomi- nal Operations Upon the Pelvic Genital Organs, the Selection and Mortality Experience of, C. T. Brown	VI	31
Discussion—Beckett and Porter	VI	42
Ferguson, John. Epilepsy in Relation to Life In- surance	IX	87
Ferguson, John. Total and Permanent Disability Benefits for Women	XV	302
Finney, J. M. T. Surgery of the Digestive Organs	XVI	203-218
Discussion—Ill, Cragin, Hutchinson, Finney	XVI	219-233

	Volume	Page
Fisher, John W. Goitre	XV	58
Fisher, J. W.		
Report of the Committee on Blood Pressure	VII	17
Report of the Northwestern's Experience with Re- spect to Habits of Insured Persons in the Use of Alcoholic Stimulants—issues 1901-1908	IX	273
Fitz, Reginald. Reference to His Work on Rapidity of Absorption from the Stomach of Glucose Solution	XVI	199
Flarimeter	XVI 41, 44, 68	
Abnormal Tests on 88 Normal Males. Table 12..	XVI	82
Calibration of First 18 B-D Flarimeters	XVI	73
Comparative Tests on 9 Prudential Female Track Athletes	XVI	76
Comparative Tests on 22 Prudential Male Track Athletes. (Table)	XVI	75
Directions for Applying Functional Test	XVI	102
Normal Lengths of Blow with Large Orifice by Height. Adult Males	XVI	104
Temperature Effect in Flarimeter No. 17 (Room Temperature 26° c)	XVI	71
Tested by Dr. Bernard A. Schwartz in Heart Clinic	XVI	132
Tests on 88 Normal Adult Males Table II	XVI	81
Wells Demonstrates Use of Flarimeter	XVI	113
Flarimeter Test. Time Requisite for Performance..	XVI	97
Fluroscope. Value in Life Insurance Examination	XVI	267
Focal Infections in Relation to Life Insurance. H.		
B. Anderson	IX	144
Discussion—Fraser, Wilson, Exton, Hobbs..	IX	144
Focal Infection, in Relation to Life Insurance.		
W. M. Bradshaw	VII	232
Discussion—Porter, Wilson, MacKenzie, Martin, Chapin	VII	232

Cumulative Index

407

	Volume	Page
Folin, O. H.		
Metropolitan Laboratories	x	111
Some New Observations on the Distribution of Sugar Within the Animal Body	xiv	428
Folin, Otto and Normon R. Blatherwick. Blood Sugar Curves After the Ingestion of 50 Grams of Glucose	xvi	155
Discussion—Benedict, Cort, McCrudden	xvi	183-197
Fraser, F. R. Summary of Goulstonian Lectures on Cardiac Dyspnea	xvi	93
Fraser, Robert A. Report of Availability of Sta- tistics and Present Status of Information as to Gassing and Shell Shock	vi	74
French Aviation Service	xvi	48
Frost, Harold M.		
The Cardio-Respiratory Test: Analysis of Expe- rience for the Past Five Years	xiv	183
Report on Cardio-Respiratory Test	xii	62
Report on Progress of the Application of the Car- dio-Respiratory Test	x	232
A Study of Cardio Vascular Reaction to Abnor- mal Variations of Intrathoracic Pressure	ix	207
Frost, Harold M.		
Discussion of Paper on Functional Tests of the Circulation	xvi	123
Functional Tests of the Circulation. L. F. Mac- Kenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	xvi	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	xvi	114-135
Bibliography	xvi	111-113
Directions for Applying	xvi	102
Gall Bladder Disease	xvi	248-262
The Liver and Pancreas in Gall Bladder Infection	xvi	250
Mortality Tables Gall Stones and Biliary Colic	xvi	254

	Volume	Page
Gall Bladder Patient, Outlook for Life and Health of, F. S. Mathews	XIII	139
Discussion—Rowley and Fisher	XIII	160
Gassing and Shell Shock, Report of Availability of Statistics and Present Status of Information as to, R. A. Fraser	VI	74
Discussion—Strathy, Archibald, Martin, An- derson, McCulloch and Whitney	VI	83
Gastric Ulcers	XVI	220
Mortality on Substandard and Underwriting— Cases of Gastric and Duodenal Ulcer	XVI	227
Operative Mortality Rate	XVI	218
Study of 510 Cases of Gastric and Duodenal Ul- cer, 1900-1925, Johns Hopkins and Union Me- morial Hospitals Baltimore Summarized by Finney		209-218
Tables Showing History of Individuals with Gas- tric and Duodenal Cured Ulcers to Whom In- surance Policies were Issued. Records Extend Over Period of 18 Years	XVI	221-223
Gastro-Intestinal Tract.		
Bibliography on Medical Aspect of Gastro-Intes- tinal Diseases	XVI	252
Colitis	XVI	246
Incidence of Gastro-Intestinal Disease in General Population Group.	XVI	265
Medical Aspects of Certain Gastro-Intestinal Tract Disease. W. F. Hamilton	XVI	236
Operative Mortality	XVI	214
Ratio of Deaths from Intestinal and Peritoneal Tuberculosis to That of Total Deaths from Tu- berculosis in All Forms in Canada, 1922-1927..	XVI	277
Study of 510 Cases of Gastric and Duodenal Ul- cer, 1900-1925, Johns Hospkins and Union Me-		

Cumulative Index

409

	Volume	Page
morial Hospitals, Baltimore—Summarized by Finney	xvi	209-218
Surgery of the Digestive Organs. J. M. T. Finney	xvi	203-218
Tuberculosis of the Gastro-Intestinal Tract. J. F. Honsberger	xvi	270
Tuberculosis of Intestine and Peritoneum	xvi	244
Gaylord, Harvey R. Address on Cancers	viii	231
Gilchrist, Ralph T. Presentation of Dr. Fisher's Paper on Goitre	xv	58
Glycosuria.		
Advantages of Rate of Excretion Over Concen- tration as the Criterion in Proteinuria and Gly- cosuria. William G. Exton and Anton R. Rose	xvi	143
Discussion—Benedict, Cort, Brown, Mc- Crudden, Exton	xvi	184-198
Bibliography of Recent Literature. S. J. Streight..	xv	97-99
Charts. Frederick G. Braithwaite	xv	100-107
The Clinical Significance of Intermittent Glycos- uria from the Viewpoint of Insurance Medi- cine. William M. Bradshaw and Frank H. Carber	xv	66
Discussion—Mann, Folin, Bradshaw, Wolf, Knight, Shewbrooks, Cort, Dillon, Exton, Streight	xv	107-140
Its Importance in Life Insurance Selection. R. Huston	x	205
Discussion — Truitt, McCloud, Strathy, Daley, Exton, Muhlberg and Brown	x	205
Preliminary Communication of a Method of Es- timating the Significance of, W. G. Exton	ix	258
Discussion—Blakely, Pauli and Bradshaw	ix	258
A Proposed Method of Selecting Risks Among Individuals with Occasional Slight, F. H. Mc- Crudden	xi	156

	Volume	Page
Discussion — Benedict, Rolph, Bradshaw, Dwight, Folin and Rockwell	XI	229
The Study of Low Threshold and Other Non- Diabetic Types of Glycosuria in Applicants for Life Assurance. S. J. Streight	XV	79
Goitre. John W. Fisher	XV	58
Goitre, The Present Status of, F. Carber	XI	270
Discussion—Hutchinson, Bradshaw, Pollard and Russell	XI	281
Goitre, Remarks on, H. C. Scadding	VIII	269
Goitre, Report of the Joint Committee on Mortality Among Persons with	XII	20
Goitre, Report on, G. A. Harlow	VIII	265
Gore, John K. Remarks to Convention	XVI	10
Graphic Standard Table. O. H. Rogers	XIII	44
Graves, William W. Age Incidence of Scapular Types, Its Possible Relation to Longevity	X	60
Grosvenor, Frank L.		
Experience on Certain Border-Line Classifications	X	6
Group Insurance, Its Medical Aspects	VII	167
Group Insurance, Its Medical Aspects	VII	172
Discussion—Willard, Wells, Root, Fitzger- ald, Cook	VII	172
Mitral Insufficiency—A Limited Experience, In- cluding Etiology	XIII	199
Habits of Insured Persons in the Use of Alcoholic Stimulants Issued in 1901-1908, Experience with Respect to, J. W. Fisher	IX	273
Hamilton, W. F. Medical Aspects of Certain Gas- tro-Intestinal Tract Diseases	XVI	236
Discussion—Wenstrand, Russell, Fellows.....	XVI	253-263
Hanrahan, E. M. Reference to His Study of 510 Cases of Gastric and Duodenal Ulcer, 1900- 1925	XVI	209-218
Hardin, John R. Address of Welcome	XI	5

Cumulative Index

411

	Volume	Page
Harlow, George A. Report on Goitre	VIII	265
Harnden, Frank Discussion of Paper on Disability and Digestive Disorders	XVI	298
Hart, T. Stuart. The Significance of Heart Mur- murs and Irregularities	XI	107
Health Examinations for Policyholders, The Value of Periodic, H. Toulmin	XI	47
Discussion—Knight, Pauli, Geiringer, Fisk and Honsberger	XI	51
Heart.		
Flarimeter Tested by Dr. Bernard A. Schwartz in Heart Clinic	XVI	132
Functional Tests of the Circulation. L. F. Mac- Kenzie, P. V. Wells, E. G. Dewis, L. D. Ylvisaker	XVI	36
Summary of Professor F. R. Fraser's Gouls- tonian Lectures on Cardiac Dyspnea	XVI	93
Ventricular Fibrillation. Yandell Henderson	XVI	117
Heart Disease.		
The Abnormal Pulse, Its Detection, Recognition and Evaluation. Gordon Wilson	XV	192
Discussion—Cook, Rogers, Old, Cragin	XV	218
William Muhlberg	XV	113
Experience on Risks with Mitral Regurgitation.		
T. H. Rockwell	IX	173
Discussion—Russell, Symonds, Eakins, Chapin	IX	173
Heart Murmurs—Their Influence on Longevity.		
O. H. Rogers and A. Hunter	VI	173
Myocardial Degeneration and Coronary Throm- bosis. Alexander Lambert	XV	200
Discussion—Cook, Rogers, Old, Wilson, Cra- gin, McDonald	XV	218-245
The Significance of Heart Murmurs and Irregu- larities. T. S. Hart	XI	107

	Volume	Page
Discussion—Root, Hart, Sykes, Baker, McCrudden, MacKenzie	XI	120
Heart, Electrocardiography. Cardiography and Oscillometry in Life Insurance Selection. Frederick G. Brathwaite	XVI	19
Height and Weight.		
Chest and Abdominal Measurements as Related to Height and Weight, with Presentation of Tables of Averages. Charles P. Clark	XVI	341
Chest and Abdominal Measurements as Related to Height and Weight. (Statistical Study) John S. Thompson	XVI	352
Discussion—Weisse, Piper	XVI	361-362
Henderson, R. Relationship with Actuary	XV	35
Henderson, Robert and T. H. Rockwell. Effect on Mortality of a History of Tuberculosis	XIV	396
Henderson, Yandell. Discussion of Paper on Functional Tests of the Circulation	XVI	116-123
Hobbs, A. B.		
Discussion of Paper on Women as Life Insurance Risks	XVI	330
Syphilis—A Study	XIII	14
Honorary Members of the Association	XIV	459
Honsberger, Jerome F. Tuberculosis of the Gastro-Intestinal Tract	XVI	270
Discussion—Ward, Van Dervoort	XVI	278-280
Hunter, Arthur.		
The Influence of Occupation Upon Mortality	VII	88
Is the Average the Same as the Normal for Weight and Blood Pressure?	VII	149
Numerical Methods of Determining the Value of Risks for Life Insurance	VI	99
Heart Murmurs—Their Influence on Longevity	VI	173

Cumulative Index

413

	Volume	Page
Blood Pressure ,as Affected by Sex, Weight, Climate, Altitude, Latitude, or by Abstinence from Alcoholic Beverages	VI	92
Ratings for the Principal Impairments	VIII	121
Hunter, Arthur and O. H. Rogers.		
A Mortality Study of Impaired Lives, (a) Inflammatory Rheumatism, (b) Tuberculosis of Lungs and Blood Spitting	IX	161
Discussion—Russell, Symonds, Eakin and Chapin	IX	161
Mortality Study of Impaired Lives — x, 43; xi, 96; xii, 71; xiii, 176; xiv, 54; xv, 412		
Discussion—Patton, Bradshaw, Exton and Clark	xii	84
Discussion—Fisher, Eakins and Frost	xiii	186
Discussion—Weisse, Muhlberg, Rockwell and Wilson	xiv	62
Huston, Ross.		
Glycosuria, Its Importance in Life Insurance Selection	x	205
Practical Methods for Promoting the Consideration of Applications by Lay Approvers	xi	145
Hutchinson, James P. Discussion of Paper on Surgery of Digestive Organs	xvi	230
Ill, Edward J. Discussion of Paper on Surgery of Digestive Organs	xvi	219
Impaired Lives, Mortality Study of (a) Inflammatory Rheumatism, (b) Tuberculosis of Lungs and Blood Spitting. A. Hunter and O. H. Rogers	IX	161
Discussion—Russell, Symonds, Eakins, Chapin	IX	161
Impaired Lives, Mortality Study of. O. H. Rogers and A. Hunter	x, 43; xi, 96; xii, 71; xiii, 176; xiv, 54; xv, 412	

	Volume	Page
Discussion—Patton, Bradshaw, Exton and Clark	XII	84
Discussion—Fisher, Eakins and Frost	XIII	186
Discussion—Weisse, Muhlberg, Rockwell and Wilson	XIV	62
Resolution to Undertake New Mortality Investigation of Medical Impairments. O. Rogers	xv	53
Discussion—Rockwell, Daley	xv	53-54
Impaired Risks, an Endeavor on the Part of Canadian Companies to Secure Greater Uniformity in the Treatment of, H. C. Scadding	ix	229
Discussion—Hamilton, Archibald, Patton	ix	229
Impairments Arising as a Result of Military Service. G. S. Strathy	vi	250
Influence (The) of Occupation Upon Mortality. O. H. Rogers and A. Hunter	vii	125
Discussion—Foshay, Murphy, Dublin, Fitzgerald	vii	125
Influenza, Notes on the Likelihood of a Recurrence of, H. Toulmin	vi	52
Discussion—Fisher, Rowley, Symonds, Anderson	vi	58
Influenza, Report of the Commission of the Metropolitan Life Insurance Company. M. J. Rosenau	viii	62
Discussion—Symonds, McMahon, Weisse and Willard	viii	62
Insurance Record Affect Selection? Does the. T. H. Rockwell	xiii	67
Discussion—Van Kleeck, Pollard, Eakins and Fisher	xiii	72
Is the Average the Same as the Normal for Weight and Blood Pressure? A. Hunter	vii	155
Discussion—Strong, Symonds, Fisher, MacKenzie	vii	155

Cumulative Index

415

	Volume	Page
Johann, A. E. Apical Cardiac Murmurs	XIV	155
Jones, Frank L., Address of Welcome	XV	9
Joslin, Elliott P. Diabetes and Life Insurance	VIII	87
Kingsbury, F. B.		
Demonstration of Urinary Tests	XI	252
Progress in Quantitative Laboratory Method	XIV	383
Kingsley, Darwin P. Address of Welcome	IX	6
Knight, Augustus S.		
Address of the President	VIII	6
Weight, Tables of Over and Under, Corresponding to Various Mortality Ratios	IX	193
Knight, Augustus S. and Louis I. Dublin. Mortality, Morbidity and Working Capacity of Tuberculosis Patients After Discharge from the Metropolitan Life Insurance Company Sanatorium Between 1914 and 1927	XV	247
Discussion—Ordway, Rollins, Fellows	XV	269-281
Lambert, Alexander. Myocardial Degeneration and Coronary Thrombosis	XV	200
Discussion—Cook, Rogers, Old, Wilson, Cragin, McDonald		218-245
Life Insurance Selection.		
Cardiography Oscillometry in Life Insurance Selection. Frederick G. Brathwaite	XVI	19
Life Insurance Selection, J. Allen Patton	XVI	11
Women as Life Insurance Risks. Wendell M. Strong and Faneuil A. Weisse	XVI	307
Life Insurance Without Medical Examination.		
H. C. Scadding	XII	223
Discussion—Rowley, Piper, Archibald, McCulloch, Jenney, Hamilton and Hobbs	XII	243
List of Members	XV	427
	XVI	369
Liver and Pancreas in Gall Bladder Infection	XVI	250
Low, Ethelbert I. Address of Welcome	XII	4

	Volume	Page
Lowsley, O. S. Urologic Surgery	XII	107
McCall, James C. Address of Welcome	XIII	76
McCrudden, Francis H. A Proposed Method of Selecting Risks Among Individuals with Occa- sional Slight Glycosuria	XI	156
MacKenzie, L. F. Functional Tests of the Circula- tion. L. F. MacKenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	XVI	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	XVI	114-135
MacKenzie, L. F. and J. A. Patton. The Pulse in Life Insurance	XIII	221
McMahon, Thos. F. Address of the President	IX	9
MacSweeney, Edward S. Disability and Mortality in Arrested Tubercular Cases	XII	142
Martin, C. F. Pleurisy in Relation to Life Insur- ance	X	182
Master and Oppenheimer's Table	XVI	59
Mathews, Frank S. Outlook for Life and Health of the Gall Bladder Patient	XIII	139
Medical Aspects of Certain Gastro-Intestinal Tract Diseases. W. F. Hamilton	XVI	236
Discussion—Wenstrand, Russell, Fellows ..	XVI	253-263
Medical Directors. O. M. Eakins	X	128
Discussion—Willard, Symonds and Fisher ..	X	128
Medical Directors:		
Relationships of Medical Directors. R. M. Daley ..	XV	12
Relationship with Examiners. L. G. Sykes	XV	17
Relationship with Actuary. R. Henderson	XV	35
Relationship with Executives. H. Moir	XV	42
Relationship with the Association. R. M. Daley ..	XV	46
Relationship with Each Other and Insurance in General. O. Rogers	XV	49

Cumulative Index

417

	Volume	Page
Medical Examiners:		
Committee to Study and Report on Advisability of Educating and Listing Examiners	xv	190
Relationship with Examiners. L. G. Sykes	xv	17
Discussion—Wilson, Wells, McDonald, Huston, Root, Patton, Righter, Alton	xv	25-30
Medical Examiners for Life Insurance. F. L. Wells	xii	121
Discussion—Beckett, Grosvenor, Muhlberg, Russel, Toulmin, Milroy and Wilson	xii	129
Medical Impairments: Resolution to Undertake New Mortality Investigation of Medical Impairments. O. Rogers	xv	53
Discussion—Rockwell, Daley	xv	53-54
Members of the Association of Life Insurance Medical Directors, List of ..vi, 257; vii, 361; viii, 313; ix, 283; x, 287; xi, 347; xii, 295; xiii, 297; xiv, 445; xv, 427; xvi, 369		
Membership	xvi	369
Candidates for	xvi	2
Emeritus Members	xv, 4; xvi,	379
Honorary Members	xvi	379
List of Members	xv, 427; xvi,	369
New Members Elected	xv, 3; xvi,	3
Memorandum. J. W. Fisher	x	40
Metropolitan Laboratories. S. R. Benedict and O. H. Folin	x	111
Discussion—Exton and Balch	x	111
Military Service on Insurance Risks, Further Experience in Estimating the Effect of, G. C. Strathy	vii	244
Miller, James Alexander. Relative Value of the Various Factors in the Diagnosis of Suspected Tuberculosis	xi	127
"Mistakes." O. M. Eakins	xiv	46

	Volume	Page
Mitral Insufficiency—A Limited Experience, Including Etiology. F. L. Grosvenor	XIII	199
Discussion—Cook and Frost	XIII	203
Mitral Regurgitation, Experience on Risks with, T. H. Rockwell	IX	173
Moir, H. Relationship with Executives	XV	42
Morbidity: Single and Married Women Compared. Mortality.	XV	350
Mortality Among Persons with Goitre, Report of the Joint Committee on.	XII	20
Mortality Investigation of Male Lives Accepted Without Medical Examination. H. Crawford Scadding	XV	281
Discussion—Rockwell, Baker, Jaquith, Patton, Cragin, Grosvenor	XV	286-301
Mortality, Morbidity and Working Capacity of Tuberculosis Patients After Discharge from the Metropolitan Life Insurance Company Sanatorium Between 1914 and 1927. Knight, Augustus S. and Louis I. Dublin	XV	247
Discussion—Ordway, Rollins, Fellows	XV	269-281
Resolution to Undertake New Mortality Investigation of Medical Impairments in Cooperation with the Actuarial Society of America	XV	53
Discussion—Rockwell, Daley	XV	53-54
Mortality Ratings. Resolution. O. Rogers	XV	55
Discussion—Willard	XV	55
Mortality Study of Impaired Lives.		
O. H. Rogers and A. Hunterx, 43; XI, 96; XII, 71; XIII, 176; XIV, 54; XV, 412		
Discussion—Brown, Hamilton, Knight and Fisher	X	43
Discussion—Patton, Bradshaw, Extton and Clark	XII	84
Discussion—Fisher, Eakins and Frost	XIII	186

Cumulative Index

419

	Volume	Page
Discussion—Weisse, Muhlberg, Rockwell and Wilson	XIV	62
Introductory Remarks	XIII	170
Motions.		
Adoption of Minutes of Executive Council Meet- ing of May 16, 1929 and October 23, 1929	XVI	4
Expressing Appreciation of the Society for the Hospitality of the Prudential Insurance Com- pany in Entertaining the Convention, Piper.....	XVI	339
Reading of Minutes of Thirty-ninth Annual Meeting—Waived	XVI	4
Regarding Payment of Dues	XVI	10
Roll Call Waived	XVI	2
Secretary to Cast Ballot in Favor of Election of Each Candidate for Membership	XVI	4
To Appoint a Committee to Draw Up a Resolu- tion Expressing Association's Appreciation of Dr. Roger's Work	XVI	115
To Close Nomination	XVI	9
Instructing Secretary to Cast Ballot for Officers as Nominated	XVI	9
Treasurer's Report Accepted	XVI	9
Muhlberg, William.		
Conservation Work, Based on Annual Urinalysis and Policyholder's Statement of Health	VI	12
Discussion of Paper on Disability and Digestive Disorders	XVI	302
Discussion of Paper on Functional Tests of the Circulation	XVI	129
Heart Disease. Discussion	XV	113
Introductory Remarks to Paper	XVI	164
A Practical Survey of the Newer Chemical Uri- nary Tests	XIII	86
Selection of Medical Examiners for Life Insur- ance	X	260

	Volume	Page
Muhlberg, William and Jeanette Allen Behre. The Determination of Acetone in the Urine and Its Significance in Life Insurance Examinations ..	xvi	167
Discussion—Benedict, Cort, Brown	xvi	184-195
Myocardial Degeneration and Coronary Thrombo- sis. Alexander Lambert	xv	200
Discussion—Cook, Rogers, Old, Wilson, Cragin, McDonald	xv	218-245
Nominating Committee, Report ... xii, 17; xiii, 31;	xiv, 10; xv, 7; xvi, 8	
Non-Medical Factors Affecting Mortality. H. A. Baker	xii	211
Discussion—Lounsberry and Eakins	xii	216
Non-Medical Mortality: Mortality Investigation of Male Lives Accepted Without Medical Examination. H. Crawford Scadding	xv	281
Discussion—Rockwell, Baker, Jaquith, Pat- ton, Cragin, Grosvenor	xv	286-301
Numerical Method of Determining the Value of Risks for Life Insurance. O. Rogers and A. Hunter	vi	99
Discussion—Henderson, Rhodes, Hutcheson, Mead, Sheppard, Coburn and Knight, Symonds, Toulmin	vi	129
Numerical Ratings in the Selection of Risks. O. M. Eakins	vii	208
Discussion—Hall, Brown, Cook	vii	208
Obituaries: Billings, A. W.	xv	6
Burr, C. R.	xi	7
Chapin, F. W.	xvi	5
Craig, T. C.	ix	14
Eagleson, J. B.	xv	6
Emery Z. Taylor	xi	9

Cumulative Index

421

	Volume	Page
Gedge, Donald MacColloch	XIII	5
Haines, I.	XI	8
Hall, George Clifton	XII	7
Johnson, A. J.	VIII	31
Kellogg, Edward B.	XIV	9
Knight, W. W.	XI	11
Little, John M.	XIII	9
Lounsberry, Robert L.	XIII	8
Martelle, Henry A.	XVI	6
Merchant, F. D.	X	32
Miller, W. R.	X	32
Northcott, Edwin M.	XII	8
Peterson, William A.	XII	9
Porter, William Evelyn	XII	10
Post, Albert Todd	XII	12
Priestly, James T.	XIII	6
Stebbins, George S.	XII	13
Strathy, George Stewart	XII	14
Symonds, B.	XI	12
Toulmin, Harry	XVI	7
Watson, William Perry	XII	15
Webb, J. H.	VIII	29
Wells, Ernest Alden	XIV	7
Wells, F.	VI	9
Wells, F. C.	XI	14
Woolen, G. V.	IX	14
Young, John C.	XIV	8
(See Also: Deceased Members.)		
Officers.	XV	v
Officers of the Association Since Its Organization	XVI	vi
Old, Herbert. Discussion of Paper on Cardiogra- phy and Oscillometry in Life Insurance Selec- tion	XVI	31
Oscillometry	XVI	16-31

	Volume	Page
Cardiography and Oscillometry in Life Insurance		
Selection. Frederick G. Brathwaite	xvi	19
Practical Oscillometry	xvi	28
Value of Oscillometry in Life Insurance Selection	xvi	30
Outlook for Life and Health of the Gall Bladder		
Patient. F. S. Mathews	xiii	139
Discussion—Rowley and Fisher	xiii	160
Overweight.		
Chest and Abdominal Measurements as Related to Height and Weight, with Presentation of Tables of Averages, Charles P. Clark	xvi	341
Chest and Abdominal Measurements as Related to Height and Weight (Statistical Study) John S. Thompson	xvi	352
Discussion—Weisse, Piper	xvi	361-362
Overweight.		
Relation Between Overweight and Cancer—a Preliminary Examination of Evidence from Insurance Statistics. Louis I. Dublin	xv	402
Discussion—Muhlberg	xv	408
Overweights—How They Should Be Treated, on the Basis of Recent Studies	vi	216
Discussion — Fisher, McKechnie, Root, Wood, McMahon, Symonds, Gore, Nichols	vi	216
Overweights Selection and Statistics, Twenty Years of, F. S. Weiss and W. M. Strong	xii	24
Discussion — Ward, Thorndike, Jaquith, Dwight and Rogers	xii	45
“Oxygen Debt”	xvi	57
Pancreas	xvi	263
Liver and Pancreas in Gall Bladder Infection	xvi	250
Patton, J. Allen	xv	424
Report of the Committee on Urinary Impairments		xi, 244; xii, 115; xiii, 34; xiv, 12

Cumulative Index

423

	Volume	Page
Demonstration of Urinary Tests: Clark and Kinsbury	XI	250
Discussion — Cook, Rockwell, Bradshaw, Benedict and Folin	XI	253
Discussion—Dwight, Cragin and Muhlberg..	XII	116
Presidential Address	XVI	11
Patton, J. Allen and L. F. MacKenzie. The Pulse in Life Insurance	XIII	221
Paul, George P. Disability Benefits	xv	354
Discussion—Olsen, Wells, Wehner	xv	369-401
Peabody, C. A. Address of Welcome	vii	4
Peptic Ulcers	xvi	220
Bibliography	xvi	293
Cancer of the Stomach and Its Relation to Ulcer of the Stomach	xvi	241
Operative Mortality Rate	xvi	218
Results of Treatment	xvi	287
Study of 510 Cases of Gastric and Duodenal Ulcer 1900-1925, Johns Hopkins and Union Memorial Hospitals, Baltimore—Summarized by Finney	xvi	209-218
Tables Showing History of Individuals with Gastric and Duodenal Cured Ulcers to Whom Insurance Policies Were Issued. Records Extend Over Period of 18 Years	xvi	221-223
Table of Mortality on Substandard Underwriting —Cases of Gastric and Duodenal Ulcer	xvi	227
Photo-Electric Scopometer. William G. Exton	xvi	141
Discussion—Benedict, Cort, Brown, Exton ..	xvi	182-199
Piper, Charles B. Discussion of Paper on Chest and Abdominal Measurements as Related to Height and Weight	xvi	362
Pleurisy in Relation to Life Insurance. C. F. Martin	x	182

	Volume	Page
Discussion—Bartlett, Gordon, Wilson, and Ward	x	182
Practical Methods for Promoting the Consideration of Applications by Lay Approvers. R. Huston	xi	145
Discussion—Kanouze and Everett	xi	152
Practical Survey of the Newer Chemical Urinary Tests, W. Muhlberg	xiii	86
Introductory Remarks	xiii	84
Discussion—Clark, Kingsbury, and Exton ..	xiii	97
President's Addressvi, 4; vii, 6; viii, 6; ix, 9; x, 6; xi, 16; xii, 17; xiii, 12; xiv, 6		
"Pressprobe" Breath Holding Test	xvi	48
Progress in Quantitative Laboratory Method. F. B. Kingsbury	xiv	383
Discussion—Clark	xiv	392
Prohibition, if National Prohibition Becomes Effective, What Shall Be Done with People Who Give a Past History of Intemperance, or Were Recently Connected with the Sale of Liquors....	vi	229
Discussion—Beckett, Linton, Hall, Morris, Nichols, Van Wagenen, Turner, Toulmin, Watt, Marshall, Moir, Wood, Little, Gibb ..	vi	230
Proteinuria. Advantages of Rate of Excretion Over Concentration as the Clinical Criterion in Proteinuria and Glycosuria. William G. Exton and Anton R. Rose	xvi	143
Discussion—Benedict, Cort, Brown, McCrudden, Exton	xvi	182-198
Prudential Longevity Service. W. G. Exton	x	244
Discussion—Muhlberg, Daley and Patton ..	x	244
Public Health Committee of the Association of Life Insurance Medical Directors	vii	243, 244

Cumulative Index

425

	Volume	Page
Public Health, The Need of General Education in the Matter of, T. H. Willard	VII	81
Discussion—Symonds, Beckett, Patton, Wells	VII	81
Pulse in Life Insurance, J. A. Patton and L. F. MacKenzie	XIII	221
Discussion—Dwight, Cook, Wishard and Martin	XIII	260
Pulse Rate and Exercise	XVI	56
Quantitative Microscopic Urinalysis. William G. Exton	XV	146
Discussion—Patton and Clark	XV	182
Ratings for the Principal Impairments. A. Hunter and O. H. Rogers	VIII	121
Discussion—Henderson, Rockwell, Toulmin, Weisse, and Eakins	VIII	121
Reaction of the Diastolic Pressure to the Cardio-Respiratory Test. H. H. Amiral	XIV	219
Discussion—Rogers, Frost and Muhlberg	XIV	234
Reiley, Austin D. Disability Benefits—Selection from the Underwriting Standpoint	XIII	287
Relation Between Overweight and Cancer—a Preliminary Examination of Evidence from Insurance Statistics. Louis I. Dublin	XV	402
Discussion—Muhlberg	XV	408
Relationships of Medical Directors. (See: Medical Directors.)		
Reminiscences and After Dinner Talk. G. A. Van Wagner	XI	342
Renal Colic: Mortality Study of Impaired Lives. Oscar H. Rogers and Arthur Hunter. No. 7 (a) Renal Colic (b) Biliary Colic	XV	412
Report of Nominating Committee	XV	7
Report on Progress of the Application of the Cardio-Respiratory Test. H. M. Frost	X	232

	Volume	Page
Discussion — Chapin, Rowley, Blakely and Archibald	x	232
Representatives: Companies and Their Representatives	xv	442
	xvi	380
Resolution Requesting Joint Committee on Mortality to Make a Systematic Survey of the Procedures in Use in This Country and Canada in the Selection of Risks, Prepare Mortality Ratings as Guides in Selection of Risks, etc., O. Rogers	xv	55
Discussion—Willard	xv	55
Resolution to Undertake New Mortality Investigation of Medical Impairments in Cooperation with Actuarial Society of America. O. Rogers	xv	53
Discussion—Rockwell, Daley	xv	53-54
Respiratory Test	xvi	52
Rheumatism. T. H. Rockwell	ix	179
Rheumatism, Data on, J. W. Fisher	ix	201
Rheumatism, Inflammatory. A Mortality Study of Impaired Lives. A. Hunter and O. H. Rogers	ix	161
Discussion—Russell, Symonds, Eakins, Chapin	ix	161
Rhodes, E. E. Some Non-Medical Thoughts Regarding Selection	xi	262
Rockwell, Thomas H.		
Does the Insurance Record Affect Selection?	xiii	67
Heart Murmur, Experience on Risks with Mitral Regurgitation	ix	173
Rockwell, Thomas H. and R. Henderson. Effect on Mortality of a History of Tuberculosis	xiv	396
Roentgenological (The) Selection of Latent or Non-Clinical Pulmonary Tuberculosis Lesions. L. G. Cole	vii	354

Cumulative Index

427

	Volume	Page
Rogers, Oscar H.		
Graphic Standard Table	XIII	44
The Influence of Occupation Upon Mortality	VII	88
Numerical Method of Determining the Value of Risks for Life Insurance	VI	99
Heart Murmurs—Their Influence on Longevity	VI	173
Blood Pressure as Affected by Sex, Weight, Cli- mate, Altitude, Latitude, or by Abstinence from Alcoholic Beverages	VI	92
Ratings for the Principal Impairments	VIII	121
Relationship with Each Other and Insurance in General	XV	49
Resolution Requesting Joint Committee on Mor- tality to Make Systematic Survey of Procedures Now in Use in This Country and Canada in the Selection of Risks, Prepare Mortality Ratings as Guides to Selection of Risks, etc.	XV	55
Discussion—Willard.		
Resolution to Undertake New Mortality Investi- gation of Medical Impairments in Cooperation with Actuarial Society—America	XV	53
Discussion—Rockwell, Daley	XV	53-54
Rogers, Oscar H. Discussion of Paper on Func- tional Tests of the Circulation	XVI	114
Rogers, Oscar H. and A. Hunter.		
A Mortality Study of Impaired Lives, (a) In- flammatory Rheumatism, (b) Tuberculosis of Lungs and Blood Spitting	IX	161
Discussion—Russell, Symonds, Eakins, Chapin	IX	161
Impaired Lives, Mortality Study ofx, 43; XI, 96; XII, 71; XIII, 176; XIV, 54; xv, 412		
Discussion—Patton, Bradshaw, Exton and Clark	XII	84
Discussion—Fisher, Eakins and Frost	XIII	186

	Volume	Page
Discussion—Weisse, Muhlberg, Rockwell and Wilson	XIV	62
Rose, Anton, R. Comment on Paper "Turbidity Micro-Methods for Blood Sugar"	XVI	177
Rose, Anton R. and William G. Exton. The Ad- vantages of Rate of Excretion Over Concentra- tion as the Clinical Criterion in Protenuria and Glycosuria	XVI	143
Discussion—Benedict, Cort, Brown, McCrud- den, Exton	XVI	182-198
Rose, A. R., W. G. Exton and P. V. Wells. A Sim- ple and Rapid Quantitative Test for Sugar in Urine	XIV	436
Rose, Anton R., F. Schattner and William G. Exton. Turbidity Micro Method for Blood Sugar	XVI	178
Discussion—Benedict, Cort, Brown	XVI	185-195
Rosenau, M. J. Report of the Commission of the Metropolitan Life Insurance Company on the Progress of Influenza	VIII	62
Rowley, R. L., Some Effects of Appendicitis	VII	263
Russell, Eugene F. Discussion of Paper on Medical Aspects of Certain Gastro-Intestinal Tract Dis- eases	XVI	258
Schattner, F. Anton R. Rose and William G. Exton. Turbidity Micro Method for Blood Sugar	XVI	178
Discussion—Benedict, Cort, Brown	XVI	185-195
Scholz, Samuel B. Discussion of Paper on Women as Life Insurance Risks	XVI	334
Schwartz, Bernard A. Tests Flarimeter in Heart Clinic	XVI	132
Scopometry. The Photo-Electric Scopometer. Wil- liam G. Exton	XVI	141
Discussion—Benedict, Cort, Brown, Exton	XVI	182-199
Selection of Risks: Resolution. O. Rogers	XV	55
Discussion—Willard	XV	55

Cumulative Index

429

	Volume	Page
Scadding, H. Crawford.		
Impaired Risks, Endeavor on the Part of Canadian Companies to Secure Greater Uniformity in the Treatment of	IX	229
Life Insurance Without Medical Examination.....	XII	223
Remarks on Goitre	VIII	269
A Mortality Investigation of Male Lives Accepted Without Medical Examination	XV	281
Discussion—Rockwell, Baker, Jaquith, Patton, Cragin, Grosvenor	XV	286-301
Scopometer, The. W. G. Exton	XIII	109
Discussion—Folin and Benedict	XIII	134
Selection, Does the Insurance Record Affect? T. H. Rockwell	XIII	67
Discussion—Van Kleeck, Pollard, Eakins and Fisher	XIII	72
Selection of Medical Examiners for Life Insurance. W. Muhlberg	X	260
Discussion—Weisse, Turner, Fisher and Strathy	X	260
Selection of Risks by Methods Other Than the Numerical Ratings. E. W. Dwight	VII	198
Discussion—Rogers, Symonds, Patton	VII	198
Selection, Some Non-Medical Thoughts Regarding, E. E. Rhodes	XI	262
Simple and Rapid Quantitative Test for Sugar in Urine. W. G. Exton, A. R. Rose and P. V. Wells	XIV	436
Discussion—Benedict and Exton	XIV	441
Smith, William B. and L. G. Sykes. Aviation and Life Insurance	XIV	74
Some New Observations on the Distribution of Sugar Within the Animal Body. O. Folin	XIV	428
Discussion—Benedict and Muhlberg	XIV	433

	Volume	Page
Sphygmometric Oscillometer	XVI	25
Diagram, Explanation of	XVI	26
Standardization of Medical Examination Blanks. H. K. Dillard and J. P. Chapman	VIII	169
Strathy, George, Further Experience in Estimating the Effects of Military Service on Insurance Risks	VII	244
Impairments Arising as a Result of Military Service. The above being an Address by Dr. Strathy at the dinner and unanimously voted to be printed into the Transactions	VI	250
Streight, S. J. The Study of Low Threshold and Other Non-Diabetic Types of Glycosuria in Ap- plicants for Life Assurance	xv	79
Introductory Remarks	xv	77
Strong, Wendell M. and F. S. Weisse, Twenty Years of Overweights Selection and Statistics..	xii	24
Strong, Wendell M. and Faneuil S. Weisse. Women as Life Insurance Risks	xvi	307
Discussion—Hobbs, Cullen, Scholz, Baker, Weisse	xvi	330-337
Study of Low Threshold and Other Non-Diabetic Types of Glycosuria in Applicants for Life As- surance. S. J. Streight	xv	79
Introductory Remarks	xv	77
Study of Urinary Analyses. F. R. Brown	xiv	343
Discussion—Gordon and Muhlberg	xiv	376
Sugar in the Urine, a Study of the More Common Methods Used to Detect, with Special Refer- ence to the Tests Employed in the Laboratory of the Equitable Life Assurance Society. A. S. Wolf	vii	332
Discussion—Pauli, Balch, Exton	vii	332
Surgery of the Digestive Organs. J. M. T. Finney..	xvi	203-218
Discussion—Ill, Cragin, Hutchinson, Finney..	xvi	219-233

Cumulative Index

431

	Volume	Page
Sykes, Lawrence G. Discussion of Paper on Disability and Digestive Disorders	xvi	300
Sykes, L. G. Relationship with Examiners	xv	17
Discussion—Wilson, Wells, McDonald, Huston, Root, Patton, Righter, Alton	xv	25-34
Sykes, L. G. and W. B. Smith. Aviation and Life Insurance	xiv	74
Symonds, Brandreth.		
Blood Pressure of Healthy Men and Women	ix	22
Value of the Medical Examiners' Opinion	vii	29
Syphilis—A Study. A. B. Hobbs	xiii	14
Introductory Remarks	xiii	12
Discussion—Weisse, Daley, Baker and Rogers	xiii	37
Thompson, John S.		
Chest and Abdominal Measurements as Related to Height and Weight. (Statistical Study) John S. Thompson	xvi	352
Discussion—Weisse, Piper	xvi	361-362
Total and Permanent Disability Benefits for Women.		
John Ferguson	xv	302
Toulmin, Harry	xvi	7
Toulmin, Harry T.		
Influenza, Notes on the Likelihood of a Recurrence of	vi	52
Report of the Committee to Review Questions on Medical Blanks	xii	18
The Value of Periodic Health Examinations	xi	47
Treasurer's Report	xiv	11
Tubercular Cases, Disability and Mortality in Arrested, E. S. MacSweeney	xii	142
Tuberculosis.		
Mortality, Morbidity and Working Capacity of Tuberculosis Patients After Discharge from the Metropolitan Life Insurance Company San-		

	Volume	Page
atorium Between 1914 and 1927. Augustus S. Knight, and Louis I. Dublin	xv	247
Discussion—Ordway, Rollins, Fellows	xv	269-281
Tuberculosis of the Gastro-Intestinal Tract. J. F. Honsberger	xvi	270
Tuberculosis of Intestine and Peritoneum	xvi	244
Tuberculosis. W. W. Beckett	ix	115
Discussion—Bradshaw and Knight	ix	115
Tuberculosis from an Insurance Viewpoint	xv	266
Discussion—Ordway, Rollins	xv	269-281
Tuberculosis in Relation to Death and Disability Claims	xv	246
Tuberculosis of Lungs and Blood Spitting. A Mortality Study of Impaired Lives. A. Hunter and O. H. Rogers	ix	161
Tuberculosis, Relative Value of the Various Factors in the Diagnosis of Suspected. J. A. Miller	xi	127
Tuberculosis, Mortality	xvi	278-279
Table Showing Ratio of Deaths from Intestinal and Peritoneal Tuberculosis to That of Total Deaths from Tuberculosis in All Forms in Canada, 1922-1927	xvi	277
Tuberculous Disease of Bone and Joints as Affecting Insurability. T. D. Archibald	xiv	310
Discussion—Bailey	xiv	320
Turbidity Micro Method for Blood Sugar. Anton R. Rose, F. Schattner and William G. Exton	xvi	178
Discussion—Benedict, Cort, Brown	xvi	185-195
Ulcers.		
(See: Peptic Ulcers and Gastric Ulcers.)		
Urinalysis	xvi	139-158
The Advantages of Rate of Excretion Over Concentration as the Clinical Criterion in Proteinuria and Glycosuria. William G. Exton and Anton R. Rose	xvi	143

Cumulative Index

433

	Volume	Page
The Determination of Acetone in the Urine and Its Significance in Life Insurance Examinations. Jeanette Allen Behre and William Muhlberg	xvi	167
Quantitative Microscopic Urinalysis. William G. Exton	xv	146
Discussion—Patton and Clark	xv	182
Reference to Work of Dwight on Urine of Boxers	xvi	194
Urinalysis, Some Incidentals of, W. G. Exton	vii	325
Discussion—Pauli, Balch, Wolf	vii	325
Tables, from Case Records, Illustrating Separate Effects of the Three Variables, Concentration, Urine Volume and Time	xvi	147
Urinary Impairments, Report of Committee on, J. A. Patton	xv, 244; xii, 115; xiii, 34; xiv, 12	
Demonstration of Urinary Tests. Clark and Kinsbury	xi	250
Discussion—Cook, Rockwell, Bradshaw, Benedict and Folin	xi	253
Discussion—Dwight, Cragin and Muhlberg ..	xii	116
Urinary Tests, A Practical Survey of the New Chemical. W. Muhlberg	xiii	86
Introductory Remarks	xiii	84
Discussion—Clark, Kingsbury and Exton ..	xiii	97
Urologic Surgery. O. S. Lowsley	xii	107
Slides	xii	110
Value of the Medical Examiners' Opinion. B. Symonds	vii	57
Discussion—Henderson, Thompson, Dwight, Rockwell, Toulmin, Patton	vii	57
Van Dervoort, Charles A. Discussion of Paper on Tuberculosis of the Gastro-Intestinal Tract ..	xvi	280
Van Wagner, George A. Reminiscences and After Dinner Talk	xi	342
Ventricular Fibrillation. Yandell Henderson	xvi	117

	Volume	Page
Vital Capacity	xvi, 42, 127,	136
Abnormal Tests on 88 Normal Males. Table 12 ..	xvi	82
The Flarimeter	xvi	68
Percentage of Normal Vital Capacity by Height, Men Tables	xvi	101
Percentage of Normal Vital Capacity by Height, Women Table	xvi	101
Ward, William R. Address of the President	xi	16
Ward, William R. Discussion of Paper on Tubercu- losis of the Gastro-Intestinal Tract	xvi	278
Weight, Tables of Over and Under, Corresponding to Various Mortality Ratios. A. S. Knight	ix	193
Weight.		
(See: Height and Weight.)		
Weisse, Faneuil S.		
Address of Acceptance	vi	51
President's Address	vii	6
Weisse, Faneuil S. and W. M. Strong. Twenty Years of Overweights Selection and Statistics..	xii	24
Weisse, Faneuil S. and Wendell M. Strong. Women as Life Insurance Risks	xvi	307
Discussion—Hobbs, Cullen, Scholz, Baker, Weisse	xvi	330-337
Wells, Fred L. Medical Examiners for Life Insur- ance	xii	121
Wells, P. V.		
Demonstrates Use of Flarimeter	xvi	113
Functional Tests of the Circulation. L. F. Mac- Kenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	xvi	36
Discussion — Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	xvi	114-135
Wells, P. V., W. G. Extton and A. Rose. A Simple and Rapid Quantitative Test for Sugar in Urine	xiv	441

Cumulative Index

435

	Volume	Page
Wenstrand, David E. W. Discussion of Paper on Medical Aspects of Certain Gastro-Intestinal Tract Diseases	xvi	253
Westfall, J. H. V. Address of Welcome	vi	6
Willard, Thomas H. The Need of General Education in the Matter of Public Health	vii	73
Wilson, Gordon.		
The Abnormal Pulse, Its Detection, Recognition and Evaluation	xv	192
Discussion—Cook, Rogers, Old, Cragin	xv	218
Wilson, McLeod C. Discussion of Paper on Disability and Digestive Disorders	xvi	294
Wolf, A. S. A Study of the More Common Methods Used to Detect Sugar in the Urine, with Special Reference to the Tests Employed in the Laboratory of the Equitable Life Assurance Society	vii	316
Women, Mortality.		
Tables of Mortality Experience	xvi	314-330
1. General comparison among men and women for 30 year period.		
2. Mortality by classes 1907-15, 16.		
3. Mortality by classes 1907-1922, 24.		
4. Women as a whole 1909-1918.		
1919-1927		
5. Mortality experience of 17 occupational classes 1907-1924, 25.		
6. Causes of death 1907-1922, 1924.		
Women as Life Insurance Risks. Wendell M. Strong and Faneuil S. Weisse	xvi	307
Discussion—Hobbs, Cullen, Scholz, Baker, Weisse	xvi	330-337
X-Ray, Value of in Life Insurance Examination —	xvi	266

	Volume	Page
X-Ray, The Value of, in the Diagnosis of Pulmonary Tuberculosis. L. Brown	VII	343
X-Ray Chest Examinations. K. Dunham. A Practical and Valuable Method for the Application of	IX	132
Discussion—Pauli	IX	132
Ylvisaker, L. S. Functional Tests of the Circulation.		
L. F. MacKenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	XVI	36
Discussion — Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	XVI	114-135

Index to Vol. XVI

	Page
Acetone:	
The determination of acetone in the urine and its significance in life insurance examinations. Jeanette Allen Behre and Wm. Muhlberg	167
Discussion—Benedict, Cort, Brown	184-195
Achylia gastrica	243
Address of the President. J. Allen Patton	11
Advantages of rate of excretion over concentration as the clinical criterion in proteinuria and glycosuria. William G. Exton and Anton R. Rose	143
Discussion—Benedict, Cort, Brown, McCrudden, Exton	182-198
Amendments:	
Regarding payment of dues as prescribed in Article 1, Section B, of the By-Laws	10
American Heart Association	31
American Life Convention:	
Delegates	4
Anthropometry:	
Chest and abdominal measurements as related to height and weight. (Statistical Study) John S. Thompson	352
Chest and abdominal measurements as related to height and weight, with presentation of tables of averages. Charles P. Clark	341
Appendicitis:	
Mortality rates	254
Attendance	1
Auditing Committee	9
Baker, Henry A.	
Discussion of paper on Women as Life Insurance Risks.....	336
Behre, Jeanette Allen, and Wm. Muhlberg.	
The determination of acetone in the urine and its significance in life insurance examinations	167
Discussion—Benedict, Cort, Brown	184-195
Benedict, Stanley R.	
Discussion of papers	182-185
Bibliographies:	
Functional tests of the circulation	111-113
Medical aspects of intestinal diseases	252
Peptic ulcer and biliary disease	293
Biliary tract, disease:	
Bibliography	293
Mortality tables	254
Results of treatment	286
Blatherwick, Norman R. and Otto Folin.	
Blood sugar curves after the ingestion of 50 grams of glucose	155-158
Discussion—Benedict, Cort, McCrudden	183-197

	Page
Blood, circulation:	
Functional tests of the circulation. L. F. MacKenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	114-135
Blood, sugar:	
Blood sugar curves after the ingestion of 50 grams of glucose. Otto Folin and Norman R. Blatherwick	155
Turbidity micro method for blood sugar. Anton R. Rose, F. Schattner, and William D. Exton	178
Brathwaite, Frederick G.	
Cardiography and Oscillometry in life insurance selection.....	19
Discussion—Old, Cragin	31-35
British Royal Air Force breath-holding test	48
Brown, F. R.	
Discussion of papers	192
Cancer:	
Cancer of the stomach	241
Cardiac dyspnea:	
Summary of Professor F. R. Fraser's Goulstonian Lectures....	93
Cardiography and Oscillometry in life insurance selection.	
Frederick G. Brathwaite	19
Discussion—Old, Cragin	31-35
Cardio-respiratory test	123
Chapin, Frank W.	5
Chest and abdominal measurements as related to height and weight. (Statistical study) John S. Thompson	352
Discussion—Weisse, Piper	361-362
Chest and abdominal measurements as related to height and weight, with presentation of tables of averages. Charles P. Clark	341
Discussion—Weisse, Piper	361-362
Circulation:	
Functional tests of the circulation. L. F. MacKenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	36
Bibliography	111-113
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	114-135
Clark, Charles P.	
Chest and abdominal measurements as related to height and weight with presentation of tables of averages	341
Discussion—Weisse, Piper	361-362
Colitis	246
Committees:	
Auditing	9
M. I. B.	9
Nominating committee report	8
To draft resolution in regard to Dr. Rogers	305
Companies and their representatives	380
Cook, Henry W.	
Discussion of paper on Functional Tests of the Circulation....	135
Cort, Parker M.	
Discussion of papers	185

Index

439

	Page
Cragin, D. B.	
Discussion of paper on Surgery of Digestive Organs	225
Cullen, George.	
Discussion of paper on Women as Life Insurance Risks.....	332
Deceased members	5, 389
Delegates from American Life Convention	4
Determination of acetone in the urine and its significance in life insurance examinations. Jeanette Allen Behre and William Muhlberg	167
Discussion—Benedict, Cort, Brown	184-195
Dewis, E. G.	
Functional tests of the circulation. L. F. MacKenzie, P. V. Wells, E. G. Dewis, and L. S. Ylvisaker	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	114-135
Diabetes:	
The determination of acetone in the urine and its significance in life insurance examinations. Jeanette Allen Behre and William Muhlberg	167
Discussion—Benedict, Cort, Brown	184-195
Digestive tract:	
Disability and digestive disorders. H. Dingman	285
Operative mortality	214
Surgery of the digestive organs. M. M. T. Finney.....	203-218
(See also: Dyspepsia, Peptic ulcer, Gastro-intestinal tract.)	
Dingman, H.	
Disability and digestive disorders	285
Discussion—Wilson, Harnden, Sykes, Muhlberg, Ding- man	294-304
Directions for applying functional tests	102
Disability and digestive disorders. H. Dingman	285
Discussion—Wilson, Harnden, Sykes, Muhlberg, Ding- man	294-304
Disability claims:	
Tables	296
Duodenal ulcers:	
(See: Peptic ulcers.)	
Dyspepsia	237
Analysis of 1,650 cases with gastric or dyspeptic symptoms. Blackford and Dwyer	240
Election of new members	3
Election of officers	201
Electrocardiograph	17, 18
Cardiography and oscillometry in life insurance selection. Frederick G. Brathwaite	19
Reasons for using in selection for life insurance	22
Executive council	v
Minutes of meetings of May 16, 1929 and October 23, 1929, adopted as read by the Secretary	4
Exercise test	51
Comparison of observed and calculated exercise required to delay systolic return two minutes	59
Standard number of ascents. Males	102
Standard number of ascents. Female	102

	Page
Exton, William G. and Anton R. Rose.	
The advantages of rate of excretion over concentration as the clinical criterion in proteinuria and glycosuria	143
Discussion—Benedict, Cort, Brown, McCrudden, Exton	182-198
Exton, William G.	139
The photo-electric scopometer	141
Discussion—Benedict, Cort, Brown, Exton	182-199
Exton, William G., Anton R. Rose, and F. Schattner.	
Turbidity micro method for blood sugar	178
Discussion—Benedict, Cort, Brown	185-195
Fellows, Haynes H.	
Discussion of paper on Medical Aspects of Certain Gastro- intestinal Tract Diseases	263
Finney, J. M. T.	
Surgery of the digestive organs	203-218
Discussion—Ill, Cragin, Hutchinson, Finney	219-233
Fitz, Reginald.	
Reference to his work on rapidity of absorption from the stomach of glucose solution	199
Flarimeter	41, 44, 68
Abnormal tests on 88 normal males. Table 12	82
Calibration of first 18 B-D Flarimeters	73
Comparative tests on 9 Prudential female track athletes.....	76
Comparative tests on 22 Prudential male track athletes. (Table)	75
Directions for applying functional test	102
Normal lengths of blow with large orifice by height. Adult males	104
Temperature effect in Flarimeter No. 17 (Room Tempera- ture 26°C)	71
Tested by Dr. Bernard A. Schwartz in Heart clinic	132
Tests on 88 normal adult males. Table 11	81
Wells demonstrates use of Flarimeter	113
Flarimeter test:	
Time requisite for performance	97
Fluoroscope:	
Value in life insurance examination	267
Folin, Otto and Norman R. Blatherwick.	
Blood sugar curves after the ingestion of 50 grams of glucose	155
Discussion—Benedict, Cort, McCrudden	183-197
Fraser, F. R.	
Summary of Goulstonian Lectures on cardiac dyspnea	93
French Aviation Service	48
Frost, Harold M.	
Discussion of paper on Functional Tests of the Circulation..	123
Functional tests of the circulation. L. F. MacKenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	114-135
Bibliography	111-113
Directions for applying	102

Index

441

	Page
Gall bladder disease	248-262
The liver and pancreas in gall bladder infection	250
Mortality tables:	
Gall stones and biliary colic	254
Gastric ulcers	220
Mortality on substandard underwriting—cases of gastric and duodenal ulcer	227
Operative mortality rate	218
Study of 510 cases of gastric and duodenal ulcer, 1900-1925, Johns Hopkins and Union Memorial Hospitals, Baltimore —summarized by Finney	209-218
Tables showing history of individuals with gastric and duodenal cured ulcers to whom insurance policies were issued. Records extend over period of 18 years	221-223
Gastro-intestinal tract:	
Bibliography on medical aspect of gastro-intestinal diseases..	252
Colitis	246
Incidence of gastro-intestinal disease in general population group	265
Medical aspects of certain gastro-intestinal tract disease. W. F. Hamilton	236
Operative mortality	214
Ratio of deaths from intestinal and peritoneal tuberculosis to that of total deaths from tuberculosis in all forms in Canada, 1922-1927	277
Study of 510 cases of gastric and duodenal ulcer, 1900-1925, Johns Hopkins and Union Memorial Hospitals, Baltimore —summarized by Finney	209-218
Surgery of the digestive organs. J. M. T. Finney	203-218
Tuberculosis of the gastro-intestinal tract. J. F. Honsberger	270
Tuberculosis of intestine and peritoneum	244
Glycosuria:	
Advantages of rate of excretion over concentration as the criterion in proteinuria and glycosuria. William G. Exton and Anton R. Rose	143
Discussion—Benedict, Cort, Brown, McCrudden, Exton	184-198
Gore, John K.	
Remarks to convention	10
Hamilton, W. F.	
Medical aspects of certain gastro-intestinal tract diseases.....	236
Discussion—Wenstrand, Russell, Fellows	253-263
Hanrahan, E. M.	
Reference to his study of 510 cases of gastric and duodenal ulcer. 1900-1925	209-218
Harnden, Frank.	
Discussion of paper on Disability and Digestive Disorders....	298

	Page
Heart:	
Flarimeter tested by Dr. Bernard A. Schwartz in heart clinic	132
Functional tests of the circulation. L. F. MacKenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	36
Summary of Professor F. R. Fraser's Goulstonian Lectures on cardiac dyspnea	93
Ventricular fibrillation. Yandell Henderson	117
Heart, electrocardiography:	
Cardiography and oscillography in life insurance selection. Frerderick G. Brathwaite	19
Height and Weight:	
Chest and abdominal measurements as related to height and weight, with presentation of tables of averages. Charles P. Clark	341
Chest and abdominal measurements as related to height and weight. (Statistical Study) John S. Thompson	352
Discussion—Weisse, Piper	361-362
Henderson, Yandell.	
Discussion of paper on Functional Tests of the Circulation..	116-123
Hobbs, A. B.	
Discussion of paper on Women as Life Insurance Risks.....	330
Honsberger, Jerome F.	
Tuberculosis of the gastro-intestinal tract	270
Discussion—Ward, Van Dervoort	278-280
Hutchinson, James P.	
Discussion of paper on Surgery of Digestive Organs	230
Ill, Edward J.	
Discussion of paper on Surgery of Digestive Organs	219
Life insurance selection:	
Cardiography and oscillography in life insurance selection. Frederick G. Brathwaite	19
Life insurance selection. J. Allen Patton	11
Women as life insurance risks. Wendell M. Strong and Faneuil S. Weiss	307
List of members	369
Liver and pancreas in gall bladder infection	250
MacKenzie, L. F.	
Functional tests of the circulation. L. F. MacKenzie, P. V. Wells, E. G. Dewis, L. S. Ylvisaker	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	114-135
Martelle, Henry A.	6
Master and Oppenheimer's tables	59
Medical aspects of certain gastro-intestinal tract diseases. W. F. Hamilton	236
Discussion—Wenstrand, Russell, Fellows	253-263
Membership	369
Candidates for	2
Emeritus members	379
Honorary members	379
List of members	369
New members elected	3

Index

443

	Page
Motions:	
Adoption of minutes of Executive Council Meetings of May 16, 1929 and October 23, 1929	4
Expressing appreciation of the Society for the hospitality of the Prudential Insurance Company in entertaining the convention. Piper	339
Reading of minutes of Thirty-ninth annual meeting—waived	4
Regarding payment of dues	10
Roll call waived	2
Secretary to cast ballot in favor of election of each candidate for membership	4
To appoint a committee to draw up a resolution expressing Association's appreciation of Dr. Roger's work	115
To close nomination	9
Instructing secretary to cast ballot for officers as nominated	9
Treasurers report accepted	9
Muhlberg, William.	
Discussion of paper on Disability and Digestive Disorders...	302
Discussion of paper on Functional Tests of the Circulation...	129
Introductory remarks to paper	164
Muhlberg, William and Jeanette Allen Behre.	
The determination of acetone in the urine and its significance in life insurance examinations	167
Discussion—Benedict, Cort, Brown	184-195
Nominating committee. Report	8
Obesity:	
Chest and abdominal measurements as related to height and weight, with presentation of tables of averages. Charles P. Clark	341
Chest and abdominal measurements as related to height and weight. (Statistical Study) John S. Thompson	352
Discussion—Weisse, Piper	361-362
Obituaries:	
Frank W. Chapin	5
Henry A. Martelle	6
Harry Toulalin	7
Officers	v
1928-1929	2
Election of	9, 201
Officers of the association since its organization	vi
Old, Herbert.	
Discussion of paper on Cardiography and Oscillometry in Life Insurance Selection	31
Oscillometry	16-31
Cardiography and oscillometry in life insurance selection.	
Frederick G. Brathwaite	19
Practical oscillometry	28
Value of oscillometer in life insurance selection	30
"Oxygen debt"	57
Pancreas	263
Liver and pancreas in gall bladder infection	250
Patton, J. Allen.	
Presidential address	11

	Page
Peptic ulcers	220
Bibliography	293
Cancer of the stomach and its relation to ulcer of the stomach	241
Operative mortality rate	218
Results of treatment	287
Study of 510 cases of gastric and duodenal ulcer 1900-1925, Johns Hopkins and Union Memorial Hospitals, Baltimore—summarized by Finney	209-218
Tables showing history of individuals with gastric and duodenal cured ulcers to whom insurance policies were issued. Records extend over period of 18 years	221-223
Table of mortality on substandard underwriting—cases of gastric and duodenal ulcer	227
Photo-electric scopometer. William G. Exton	141
Discussion—Benedict, Cort, Brown, Exton	182-199
Piper, Charles B.	
Discussion of paper on Chest and Abdominal Measurements as Related to Height and Weight	362
"Pressprobe" breath-holding test	48
Proteinuria:	
Advantages of rate of excretion over concentration as the clinical criterion in proteinuria and glycosuria. William G. Exton and Anton R. Rose	143
Discussion—Benedict, Cort, Brown, McCrudden, Exton	182-198
Pulse rate and exercise	56
Representatives:	
Companies and their representatives	380
Respiratory test	52
Rogers, Oscar H.	
Discussion of paper on Functional Tests of the Circulation..	114
Rose, Anton R.	
Comment on paper "Turbidity Micro-methods for Blood Sugar"	177
Rose, Anton R. and William G. Exton.	
The advantages of rate of excretion over concentration as the clinical criterion in proteinuria and glycosuria	143
Discussion—Benedict, Cort, Brown, McCrudden, Exton	182-198
Rose, Anton R., F. Schattner and William G. Exton.	
Turbidity micro method for blood sugar	178
Discussion—Benedict, Cort, Brown	185-195
Russell, Eugene F.	
Discussion of paper on Medical Aspects of Certain Gastro-intestinal Tract Diseases	258
Schattner, F., Anton R. Rose and William G. Exton.	
Turbidity micro method for blood sugar	178
Discussion—Benedict, Cort, Brown	185-195
Scholz, Samuel B.	
Discussion of paper on Women as Life Insurance Risks.....	334

	Page
Schwartz, Bernard A.	
Tests Flarimeter in heart clinic	132
Scopometry:	
The photo-electric scopometer. William G. Exton	141
Discussion—Benedict, Cort, Brown, Exton	182-199
Sphygmometric oscillometer	25
Diagram, explanation of	26
Strong, Wendell M. and Faneuil S. Weisse.	
Women as life insurance risks	307
Discussion—Hobbs, Cullen, Scholz, Baker, Weisse.....	330-337
Surgery of the digestive organs. J. M. T. Finney	203-218
Discussion—Ill, Cragin, Hutchinson, Finney	219-233
Sykes, Lawrence G.	
Discussion of paper on Disability and Digestive Disorders....	300
Thompson, John S.	
Chest and abdominal measurements as related to height and weight. (Statistical Study) John S. Thompson	352
Discussion—Weisse, Piper	361-362
Toulmin, Harry	7
Tuberculosis, mortality	278-279
Table showing ratio of deaths from intestinal and peritoneal tuberculosis to that of total deaths from tuberculosis in all forms in Canada, 1922-1927	277
Tuberculosis of the gastro-intestinal tract. J. F. Honsberger..	270
Tuberculosis of intestine and peritoneum	244
Turbidity micro method for blood sugar. Anton R. Rose, F. Schattner and William G. Exton	178
Discussion—Benedict, Cort, Brown	185-195
Ulcers:	
(See: Peptic ulcers and gastric ulcers.)	
Urinalysis	139, 158
The advantages of rate of excretion over concentration as the clinical criterion in proteinuria and glycosuria. William G. Exton and Anton R. Rose	143
The determination of acetone in the urine and its significance in life insurance examinations. Jeanette Allen Behre and William Muhlberg	167
Reference to work of Dwight on urine of boxers	194
Tables, from case records, illustrating separate effects of the three variables, concentration, urine volume and time.....	147
Van Dervoort, Charles A.	
Discussion of paper on Tuberculosis of the Gastro-Intestinal Tract	280
Ventricular fibrillation. Yandell Henderson	117
Vital capacity	42, 127, 136
Abnormal tests on 88 normal males. Table 12	82
The Flarimeter	68
Percentage of normal vital capacity by height, men. Tables	101
Percentage of normal vital capacity by height, women. Tables	101

	Page
Ward, William R.	
Discussion of paper on Tuberculosis of the Gastro-intestinal Tract	278
Weight:	
(See: Height and Weight.)	
Weisse, Faneuil S. and Wendell M. Strong. Women as life insurance risks	307
Discussion—Hobbs, Cullen, Scholz, Baker, Weisse	330-337
Wells, P. V.	
Demonstrates use of Flarimeter	113
Functional tests of the circulation. L. F. MacKenzie, P. V.	
Wells, E. G. Dewis, L. S. Ylvisaker	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	114-135
Wenstrand, David E. W.	
Discussion of paper on Medical Aspects of Certain Gastro-intestinal Tract Diseases	253
Wilson, McLeod C.	
Discussion of paper on Disability and Digestive Disorders....	294
Women, mortality:	
Tables of mortality experience	314-330
1. General comparison among men and women for 30 year period.	
2. Mortality by classes 1907-15, 16.	
3. Mortality by classes 1907-1922, 24.	
4. Women as a whole 1909-1918, 1919-1927.	
5. Mortality experience of 17 occupational classes 1907-1924, 25.	
6. Causes of death 1907-1922, 1924.	
Women as life insurance risks. Wendell M. Strong and Faneuil S. Weisse	307
Discussion—Hobbs, Cullen, Scholz, Baker, Weisse.....	330-337
X-ray, value of in life insurance examination	266
Ylvisaker, L. S.	
Functional tests of the circulation. L. F. MacKenzie, P. V.	
Wells, E. G. Dewis, L. S. Ylvisaker	36
Discussion—Rogers, Henderson, Frost, Muhlberg, Cook, MacKenzie	114-135

